CDC

MONOGRAPH

The Public Health Consequences of Disasters 1989

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES • PUBLIC HEALTH SERVICE • CENTERS FOR DISEASE CONTROL

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The Public Health Consequences of Disasters 1989

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Introduction

In June 1983, the Centers for Disease Control (CDC) was asked to write a Concept Paper entitled "Potential Public Health Threats following Major Catastrophies." It was drafted as a part of CDC's responsibilities assigned by the Principal Working Group on Health of the Emergency Mobilization Preparedness Board. Part of the Concept Paper included identification of catastrophic events associated with natural or human-generated forces of major public health consequence. Following subsequent refinement and consolidation of this list of events, CDC was asked to write a monograph that would describe the public health impact of these types of disasters.

In the report that follows, we have attempted to address this issue. Our efforts were directed at summarizing the most pertinent, recent, and useful information on these subjects—although we do not claim to have carried out a complete literature survey. Some historical review has been included to orient the reader and provide a better perspective of the subject materials. In many chapters there will be some, if not considerable, overlap of material because of the nature of the topics. This repetition serves as a reminder that some disasters significantly affect our population in diverse—yet similar and predictable—ways.

The contents of the report are divided into several major sections: General Concerns, Geophysical Events, Weather-Related Problems, and Human-Generated Problems. The first chapter, a kind of primer, describes the concepts and role of surveillance and epidemiology—the database for and the science of public health practice, respectively. The second chapter very briefly discusses some important considerations relating to communications efforts between health officials and the news media in times of disaster. The last chapter in this section addresses communicable disease control following natural disasters.

The other chapters, which cover discrete types of disasters, emphasize such areas as the history and nature of the disasters, as well as causative factors for the natural disaster that may influence morbidity and mortality. Next, we addressed the public health implications of such events including a) prevention and control measures, b) surveillance, and c) research recommendations in areas in which the public health practitioner needs more useful information. Most of the chapters follow this outline, but in some instances the format is slightly different because of the nature of the subject material. In all chapters, however, the underlying approach and the considerations taken by the authors have emphasized the level of epidemiologic knowledge of each subject. Since epidemiology is the basic science of public health, and public health directs its attention toward prevention and control of unnecessary morbidity and premature mortality, our intention has been to review what is known from an epidemiologic viewpoint and to emphasize that additional epidemiologic information is needed for a fuller comprehension of the particular problem. Woven into the content of most chapters are stresses on exposure-, disease-, or health-event surveillance, because no meaningful epidemiologic analyses or appropriate public health action can follow without reliable, objective data. It will be apparent to the reader that, with a few exceptions, epidemiologic methods have not been frequently or thoroughly applied to natural disasters and that much more information and many more analyses are needed.

Surveillance and Epidemiology

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Surveillance

Surveillance (or epidemiologic surveillance) can be thought of as the dynamic, continued scrutiny of health-related events through the systematic collection, tabulation, and analysis of relevant health data. Dissemination of this information and appropriate public health action are intrinsic to this activity. The basic functions of surveillance, therefore, are a) establishing a systematic process for collecting relevant exposure and health information, b) providing an objective and thorough analysis, and c) disseminating in a timely manner the findings and inferences of the analysis to those who need to know.

In times of national emergencies, our experiences at CDC have confirmed many times over the absolute necessity of establishing rapid surveillance systems designed to collect appropriate data and provide preliminary analyses immediately to health departments, health-care practitioners, and the public at large.

Surveillance data may relate to the health consequences of a disaster or to a disaster itself, such as a flood, hurricane, industrial or radioactive release, or deterioration in ambient air quality. In assessing the health consequences of a disaster, reported cases of disease or death serve as databases for disaster-related surveillance. However, data on laboratory tests, clinic visits, school absenteeism, or hospital admissions may be more available, current, and applicable. Sometimes, indirect measurements such as school or factory closings, counties affected, or epidemics reported may provide essential information for early assessment of a disaster. Literally any health-related measurement should be considered so that the problem can be defined. Obviously, the sensitivity and specificity of data collected must be evaluated at some time. Yet of prime concern initially is the establishment of some quantitative assessment quickly so that appropriate decisions can be made.

To determine the trends of morbidity and mortality associated with disasters, it is important to have information on the number of potential disastrous events that occur in a specified time period so that rates of injuries, disease, and death per disaster event can be generated. Monitoring certain events such as industrial and radiation releases, even when no subsequent adverse health event is apparent, is important in identifying factors leading to prevention and control measures.

Responsible surveillance also includes long-term follow-up of the population at risk. In some situations it will be important to follow exposed members of a population to determine the extent of adverse health effects associated with their exposure, e.g., individuals exposed to a chemical agent during an industrial accident.

The chapters that follow clearly illustrate how little we know of the epidemiology of certain types of disasters. This void in our knowledge attests to a major lack of surveillance systems during and immediately after crises. Although the need to establish surveillance systems may seem obvious during a major disaster, too often the public health response is intuitive rather than scientific. Mass vaccination, rapid burial of the dead, and creation of temporary hospitals represent some early responses often based on "conventional wisdom" rather than on surveillance data. Effective, timely surveillance will provide the basis for defining a health problem, establishing a priority of action, determining a strategy to prevent and control further morbidity, and eventually for evaluating the effort.

Epidemiology

Epidemiology has been defined in many ways. For our purposes it can be thought of as the study of the distribution and determinants of health-related events in human populations. So, epidemiologists study groups of people rather than single individuals.

Epidemiology is the basic science of public health practice, and consequently, the epidemiologist addresses different issues and uses special methodologies not ordinarily used by the medical practitioner. Epidemiologists traditionally attempt to answer questions such as: What agent causes disease? Where does the agent reside? How are diseases transmitted? Who is at risk of acquiring disease? What specific exposure causes disease? Control and prevention of disease in populations are the prime goals of the epidemiologist. In contrast, the physician examines a single patient, makes a diagnosis, and attempts to treat that patient.

In characterizing the health of a population—be it a school, a community, a state, or a country—the epidemiologist must know how to count cases or health events, how to analyze them objectively, and how to draw logical and defensible inferences from these analyses. The epidemiologist uses the results of these analyses to determine why people have become ill. More specifically, epidemiologic analyses provide the only scientific, objective way of determining risk factors and specific exposures directly responsible for illness and death in human populations. In practice, then, epidemiologists usually count cases of disease or death and relate these numbers to the population being studied or the population at risk. By doing so, the epidemiologist determines a rate, e.g., the number of persons with typhoid fever/100,000 persons being studied. The rate might also measure mortality, e.g., the number of deaths from drowning/100,000 persons at risk. These rates then quantitate the disease (or death) burden in a given population, which in turn allows for a valid comparison between other populations rather than simply a comparison of absolute numbers. Other rates that are determined reflect the degree of exposure to certain conditions. Then exposure rates for various populations can be compared as well.

In the case of disaster epidemiology, investigators are concerned with identifying factors that put people at increased risk of death and injury during and after the disaster event. This requires not only looking beyond the traditional epidemiologic practices described previously but also addressing such factors as land-use management, building codes, warning systems, the behavior of people during the disaster, and their willingness to comply with recommendations (e.g., evacuation).

No matter which type of rate is determined, in the final analysis the epidemiologist compares rates between different populations. If these rates differ significantly, there will usually be a biologically plausible explanation of why people have been affected. Once these risks or exposure factors are known, it is the responsibility of appropriate health officials to implement control and prevention measures.

Working with the News Media

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Although a considerable amount of material has been written on the news media and public health issues (1-4), this chapter only highlights a few key areas—since rapid communication and favorable relationships with the media during a disaster are essential components of responsible public health practice.

Most of CDC's experience relating to rapid dissemination of information and working with the media has not been associated with major natural disasters in the United States. Rather, our communications with the media, the public, and health professionals have resulted from national health crises such as the Legionnaires' disease outbreak in 1976, Guillain-Barré syndrome associated with the swine influenza immunization program in 1976, the chemical exposures in Love Canal in New York in 1979, toxic shock syndrome in 1980, and—most recently—acquired immunodeficiency syndrome (AIDS).

Nevertheless, whether for a natural disaster or a nationwide health crisis, the management of information dissemination and relationships with the media are generally the same, and some principles and guidelines are listed in the following sections.

General Guidelines

1. Health administrators and practitioners of public health need the media to disseminate important information to mutual constituents—namely, the public.

2. Knowledge of the mechanisms through which the various media operate is essential for maximizing the effectiveness of information. Newspapers, television, and radio have different methods of transmitting information and can serve to inform in unique and special ways.

3. The media represent private enterprise and are not in the business of public health. They are, therefore, driven by objectives and priorities different from those of health officials.

4. In the long run, good relationships with the news media will benefit both the public and health officials.

Specific Suggestions

1. Consider seeking the media representatives first rather than waiting for them to make contact when an emergency arises. By doing so, you can often create the agenda, control the interviews, and most importantly, place the emergency and health events in their proper perspective.

2. Appoint one individual to represent the health department or agency. Preferably this person should be a public relations or communications professional. S/he should have sole responsibility for controlling information provided to the media and public inquiries, which will minimize duplication, misinformation, and misunderstanding. All interviews with scientists and investigators should be arranged through this person.

3. Strongly consider assigning an information officer to investigators in the field so that information can be transferred rapidly from the field to central headquarters, freeing more time for the investigational team and minimizing confusion and misunderstanding.

4. Brief the information officer (and support staff) on all the scientific details of the disaster or health event on a regular basis. The more this person knows and understands, the less the need for other interviews.

5. Establish a regular media briefing time, preferably once a day (sometimes twice a day will be necessary), to coincide with press deadlines. Publicize press interviews widely, and try not to grant special interviews at any other times. Even if there is little or nothing to report, such scheduled briefings can promote better understanding.

6. Protect the scientists and technologists from interviews as much as possible. This gives them more time to work and considerably lessens an already stressful environment. Periodic interviews with the investigative team leader will be necessary to maintain a certain scientific credibility of the responsible health agency. These inter-

views can be carefully scheduled, and the scientists can be briefed in advance to smooth the interview process.

7. Coordinate all dissemination of information with state or local health departments so that all health officials have exactly the same information to give the news media. Health officials at state and federal levels should try to refer interviewers to the local health departments. The local health departments are most familiar with the situation and usually have the most to gain or lose in such circumstances.

8. Make every effort to avoid making conjecture to the media. Fill the void with facts and figures, not fantasy.

9. Instruct the person being interviewed to state at the outset what will be covered; do not allow this decision to be made by the interviewer.

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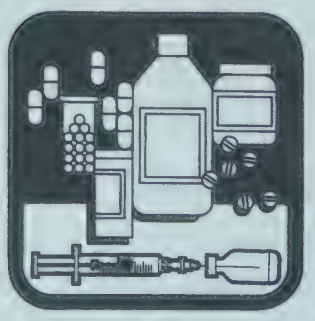

Communicable Disease Control

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Introduction

Seaman *et al.*, in their definitive chapter "Communicable Disease and Disease Control after Natural Disasters," observe that the widespread belief that epidemics of communicable disease commonly follow disasters may stem from the historical association of war, famine, and social upheavals with epidemics of smallpox, typhus, plague, and dysentery (1). In fact, during the last 40 years, outbreaks of communicable disease following natural disasters have been unusual.

In this chapter we draw heavily from our experiences at the CDC and from five other sources: the chapter by Seaman *et al.* (1), two publications by the World Health Organization (2,3), and two publications from the Pan American Health Organization (4,5).

Influences on Disease Transmission

Pathogens in the Affected Area

If the pathogen that causes a disease is not present in the affected area and not introduced after the disaster, that disease will not occur even if conditions are otherwise ideal for transmission. Smallpox has been eradicated. The areas with relapsing fever, malaria, louse-borne typhus, and plague have been greatly reduced, and are a continuing threat only where they are endemic. Seaman *et al.* (1) have concluded that the probability of a disease outbreak after a disaster is greater in developing than in developed countries. They believe that in developing countries the chief hazards are the diseases of poverty and inadequate preventive measures: diarrhea, dysentery, meningococcal meningitis, measles, malaria, intestinal parasites, pertussis, diphtheria, respiratory infections, tuberculosis, and skin diseases such as scabies.

Despite this observation, epidemiologists should be cautious in assuming that specific pathogens do not exist in an area simply because there have been no reports of disease caused by those pathogens. For example, toxigenic *Vibrio cholerae* 01 apparently persisted along the U.S. Gulf Coast for years before it was detected (6).

Movement and Density of the Population

Fortunately, large-scale movement of populations after natural disasters is relatively uncommon. When it occurs, disease transmission may be enhanced by a) an increase in the population density (facilitation of person-to-person transmission), b) the overwhelming of basic services (sanitation, water supply, vaccination), c) the contact of susceptible persons with pathogens not encountered in their home districts, and d) the introduction of diseases or disease vectors not previously found in the area. The threat of disease transmission is greatest in crowded camps. Although we know of no documented examples of the phenomenon, it is theoretically possible for relief workers to introduce diseases such as influenza that were not present in the population at the time of the disaster.

Environmental Changes

Numbers of disease vectors can increase after disasters. A rise in the number of breeding sites for mosquitoes may increase the incidence of mosquito-borne diseases such as malaria, dengue, yellow fever, St. Louis and Japanese B encephalitis, and *Wuchereria bancrofti* filariasis in areas in which the pathogens are endemic. After hurricane Flora in 1963, Haiti had an epidemic of malaria (7). Domestic flies may increase by breeding in feces, garbage, and dead animals and humans; flies may be able to transmit enteroviruses, *Shigella*, and conjunctivitis.

Disease vectors may have greater access to persons who have lost their housing (mosquitoes), are crowded together (lice), or are brought into contact with rodents (fleas). Louse-borne relapsing fever and louse-borne typhus can pose a threat in areas with a reservoir, crowded living conditions, and heavy infestations with lice, but there are currently only a few such areas worldwide. Other diseases spread by arthropods—such as leishmaniasis and murine and scrub typhus—are unlikely to occur as outbreaks after natural disasters. Numbers of dog bites were observed to increase after an earthquake in Guatemala in 1976, so rabies may be a concern, but primarily in areas in which domestic animals are the principal reservoir of the virus.

Flood waters may spread the organisms that cause leptospirosis, typhoid fever, and a host of other potentially waterborne diseases. However, their effect, if any, is apparently more likely to be exerted through contamination of water supplies than through direct infection of persons by flood waters. Leptospirosis, which can be transmitted by contact of skin or mucous membranes with contaminated water, appears to be an exception. Seaman *et al.* (1) cite two examples of outbreaks of leptospirosis following floods. One outbreak occurred in Portugal in 1967 (number of cases not reported), and the other in Brazil in 1975 (107 recorded cases).

Loss of Utilities

Failure of public water supplies, sewage systems, and power supplies that are needed to maintain the water and sewage systems can contribute to disease transmission. Water supplies are particularly crucial. Discontinuation of water service may lead persons to turn to unsafe sources of water. The lack of water can contribute to a deterioration in personal hygiene. Contamination of a large municipal system through breaks in the line, decreased pressure that allows sewage to enter the line, or disruption of water treatment can lead to the rapid transmission of pathogens to large numbers of persons. One example that was not related to a natural disaster occurred in Sangli Town, Maharashtra State, India, in 1975 and 1976, when an estimated 9,000 cases of typhoid fever followed the failure of a municipal water-treatment system (8).

Well-documented instances of waterborne disease following disasters are unusual. One reason is that the danger is usually well recognized, and the provision of safe water is almost always a top priority after a disaster. Another reason is that many areas affected by disasters have no large municipal piped water supplies; rather, wells, streams, and springs are the prime water sources and usually serve relatively small numbers of people. These small water sources are unlikely to suffer from additional contamination by human excrement after a disaster, and even when a source is contaminated only a few persons are likely to be infected. However, in at least one exception a disaster was followed by contamination of several small water sources, and an outbreak of the disease did occur. In 1971 an outbreak of 110 cases of balantidiasis (a disease caused by *Balantidium coli*, an intestinal protozoan whose principal natural reservoir is swine) followed a typhoon in Truk District, Trust Territories of the Pacific. Investigation by CDC suggested that the typhoon's disruption of catchment water supplies forced people to use many different sources of groundwater that were heavily contaminated by pig feces.

Only two post-disaster outbreaks of waterborne disease caused by contaminated municipal water supplies have been well documented during the last 40 years. In one instance, a small outbreak (23 cases) of typhoid fever occurred in Ponce, Puerto Rico, in 1956 after hurricane Betsy struck the island (9). In the other case, after a hurricane struck Mauritius in 1980, flooding allowed contaminated flood waters to back up through open taps into a low-pressure portion of the capital city's water pipes, and over 90 cases of typhoid fever resulted in that part of the city (10).

In general, in economically developed areas, prolonged disruption of basic services may increase the risk of waterborne and foodborne disease, while in areas with no piped water supplies, no electric power, and promiscuous defecation habits before the disaster, little or no increased risk of communicable diseases ensues afterwards (4).

Loss of Routine Public Health Services

After disasters, routine public health services are often disrupted by the direct effects of the disaster and sometimes by ill-conceived attempts to divert the available health resources into emergency relief programs. Mass vaccination against typhoid fever and decontamination and disposal of the dead are pernicious examples. In developed countries, vector-borne and vaccine-preventable communicable diseases may be so well controlled that interruption in routine vector control and vaccination programs against measles, pertussis, and diphtheria will have little effect on the transmission of communicable diseases. However, in developing countries a precarious balance may exist between level of disease and the effect of preventive programs. Even a brief interruption of the programs may be sufficient to give the pathogens an opportunity to spread rapidly. The hazard may be increased by other post-disaster conditions, such as increased mosquito breeding sites, population movements, and increased population density. The longer the disruption of basic public health services, the greater the risk of communicable disease.

Emergency Medical Care

When a disaster has caused traumatic injuries, there may be increased use of blood transfusions, with an attendant risk of transmission of infectious agents by blood, including hepatitis viruses and human immunodefficiency viruses (HIV). The magnitude of this risk depends on the prevalence of such infections in the donor population and the quality of screening of donors and donated blood. In fact, blood transfusions are usually given only to a small number of persons who constitute a very small proportion of the persons affected by the disaster, and well-documented instances of transfusion-related infectious disease following disasters are rare.

After disasters, increased use of injectable vaccines or medications may pose a risk of transmission of infection (e.g., hepatitis, HIV infection, and clostridial infection) by contaminated needles and syringes. Lack of adequate supplies may lead relief workers to use the same needle to inject multiple persons, and adequate disinfection of equipment may be difficult. Avoidance of unnecessary vaccination and parenteral medications combined with appropriate use of single-dose disposable needles and syringes can eliminate this potential hazard.

Theoretical Disaster-Specific Risks of Communicable Diseases

The various types of natural disasters that have occurred over the last 40 years have not usually been followed by outbreaks of infectious disease. Review of the long list of disasters in which CDC has rendered assistance (Table 1) shows none with an important and well-documented increase in the incidence of communicable disease: Truk had

TABLE 1. Outbreaks of communicable disease attributable to disasters detected in post-disaster investigations by the Centers for Disease Control, 1970-1985*

Year	Country/State	Disaster	Outbreaks
1970	Peru	Earthquake	None
	U.S.A. (Texas)	Hurricane	None
1971	Truk District	Typhoon	Balantidiasis
1972	U.S.A (S. Dakota)	Flood	None
	U.S.A. (Pennsylvania)	Flood	None
	Nicaragua	Earthquake	None
1973	Pakistan	Flood	None
1974	Sahel (W. Africa)	Famine	None
1976	Guatemala	Earthquake	None
1978	Zaire	Famine	None
	U.S.A. (Texas, Oklahoma)	Tornado	None
	Trinidad	Volcanic eruption	None
	Dominica	Hurricane	None
	Marshall Islands	Flood illness	Respiratory
1980	Marshall Islands	Typhoon	None
	Mauritius	Cyclone	Typhoid fever
	U.S.A. (Washington)	Volcanic eruption	None
	U.S.A. (multiple states)	Heat wave	None
	U.S.A. (Texas)	Hurricane	None
1982	Chad	Famine	None
	U.S.A. (Illinois)	Tornado	None
1983	Bolivia	Flood	None
1984	Mauritania	Famine	None
	Mozambique	Famine	None
	Bolivia	Famine	None
1985	Puerto Rico	Flood	None
	Colombia	Volcanic eruption	None †

*Excludes snow disasters.
† Wound infections occurred, but no outbreaks.

Sources: Ms. Nancy Nay, M.P.H., International Health Program Office, Centers for Disease Control.
Ms. Janis Videtto, Epidemiology Program Office, Centers for Disease Control.

an outbreak of balantidiasis after a typhoon in 1971, Mauritius had a small outbreak of typhoid fever after a cyclone in 1980, and the Marshall Islands had an apparent increase in mild respiratory disease after floods in 1979 forced 6,000-7,000 persons to seek temporary housing. Hence, we have few data to use in determining which communicable diseases are most likely to be associated with each type of disaster. However, we have used the available data along with our knowledge of the effects of the various types of disasters and their theoretical impact on communicable disease transmission to construct a list of communicable diseases most likely to be encountered after each type of disaster (Table 2).

Public Health Implications

Inappropriate Control Measures

After a disaster there is considerable pressure on political leaders and public health officials to take action to control communicable disease. This pressure comes from the public, the news media, overseas volunteers, and from the group of officials themselves—the leaders want to do something to help. Unfortunately, too often their actions are governed by instinctive responses rather than by a rational synthesis of available data. Usually political leaders and public health officials have had no experience in dealing with disasters, and their perceptions about the dangers they face and what should be done are the same as those of the public. They believe that communicable disease is a major threat, and in previous disasters around the world the media usually have described mass vaccination campaigns against typhoid fever and cholera and have shown photographs of masked workers spreading chemicals on corpses before burial takes place on an urgent basis. These activities are relatively easy to organize, they are highly visible and photogenic, they give health authorities a sense of accomplishment, and they generally have the vigorous approval of the population and the news media.

In reality, corpses of previously healthy people do not harbor dangerous pathogens. They pose no threat to the living other than being aesthetically disagreeable and possibly contributing to a larger fly population.

The issue of mass vaccination campaigns against typhoid fever and cholera is more complicated. After a disaster the possibility of contamination of food and water with human excrement and thus the danger of typhoid fever and cholera

TABLE 2. Theoretical risk of communicable disease, by disaster type

Disaster	Type of communicable disease transmission			
	Person-to-person*	Waterborne†	Foodborne§	Vectorborne¶
Earthquake	Medium	Medium	Medium	Low
Volcanic eruption	Medium	Medium	Medium	Low
Hurricane	Medium	High	Medium	High
Tornado	Low	Low	Low	Low
Heat wave	Low	Low	Low	Low
Cold wave	Low	Low	Low	Low
Flood	Medium	High	Medium	High
Famine	High	Medium	Medium	Medium
Air pollution	Low	Low	Low	Low
Industrial accident	Low	Low	Low	Low
Fire	Low	Low	Low	Low
Radiation	Low	Low	Low	Low

*Shigellosis, streptococcal skin infections, scabies, infectious hepatitis, pertussis, measles, diphtheria, influenza, tuberculosis, other respiratory infections, giardiasis, AIDS, meningococcal meningitis, venereal diseases, pneumonic plague.

† Typhoid and paratyphoid fevers, cholera, "sewage poisoning," leptospirosis, infectious hepatitis, shigellosis, campylobacteriosis, Norwalk agent, salmonellosis, *Escherichia coli* (enterotoxigenic, enteroinvasive, and enteropathogenic), amebiasis, giardiasis, cryptosporidiosis.

§Typhoid and paratyphoid fevers, cholera, "food poisoning," infectious hepatitis, shigellosis, campylobacteriosis, salmonellosis, *E. coli* (enterohemorrhagic, enterotoxigenic, enteroinvasive, and enteropathogenic), amebiasis, giardiasis, cryptosporidiosis.

¶Louse-borne typhus, plague, relapsing fever, malaria, viral encephalitides.

may well be greater. However, mass vaccination campaigns against these diseases are not indicated after disasters for the following reasons (11):

1. If the organism is not present in the area and is not introduced after the disaster, the disease poses no threat at all regardless of conditions; thus, in South America cholera need not be a concern. If the organism is uncommon in the area (e.g., typhoid fever in the United States), it is highly unlikely to pose a problem even if water supplies are contaminated by sewage.

2. The most practical strategy to prevent waterborne cholera or typhoid is to advise the population to boil the water before drinking it or to take other appropriate measures to ensure a safe supply of drinking water. Only this approach provides immediate protection against typhoid fever, cholera, and other waterborne disease.

3. A massive vaccination campaign cannot provide protection against typhoid at the time of greatest risk from contaminated water because a) immunity does not develop immediately and b) a second dose should be given 1-4 or more weeks later.

4. Even for persons who have received a complete series of vaccine, the typhoid vaccine is only 70%-90% effective in preventing disease, and the cholera vaccine is only about 50% effective. Neither vaccine offers important protection against spread of disease.

5. Receiving a dose of vaccine may give persons in the disaster zone a false sense of security and lead them to fail to take elementary precautions, such as boiling water.

6. Adverse reactions to cholera vaccine are not infrequent, and adverse reactions to typhoid vaccine, which are often severe, only add to the misery of the affected population. It has been observed that the only epidemics that frequently follow disasters are epidemics of sore arms and malaise reported by vaccine recipients.

7. Mass typhoid and cholera vaccination campaigns after disasters represent an unnecessary expenditure of scarce emergency health resources (personnel, transportation, money) that could be better used elsewhere.

Appropriate Control Measures

Appropriate measures to prevent and control communicable disease after a disaster include sanitary measures (emergency sanitation, provision of clean water, and vector control) (12), medical measures (vaccination and laboratory services), and a surveillance system.

Sanitary measures. Provision of clean drinking water should be a top priority. Disposal of excreta is also important. Other potentially useful sanitary measures are promotion of personal hygiene (chiefly by providing water and facilities for washing, cleaning, and bathing), food protection, and vector control (13). Efforts to control vectors should be guided by knowledge about which diseases exist in the area; often mosquitoes and lice are the primary targets, because flies and rodents are much more difficult to control and present less of a health hazard. In planning post-disaster emergency sanitation measures, relief workers should try to restore the pre-disaster levels of environmental services rather than attempting to improve on the original levels.

Medical measures. Medical measures should usually play a very minor role in preventing and controlling communicable diseases after a disaster. Although vaccination is often given undue emphasis, in areas densely populated with young children—such as refugee camps—vaccination programs against measles, pertussis, diphtheria, and poliomyelitis may be justified if the children are expected to remain there for a month or longer. Tetanus has not been common after disasters, but tetanus toxoid boosters may be indicated for previously vaccinated persons who sustain open wounds. Passive vaccination with antitoxin is useful in treating persons with wounds who have not been actively

vaccinated, as well as in treating persons with tetanus. Gas gangrene was an important problem (approximately 30 cases) for persons with deep penetrating wounds, avulsions, open fractures, and crush injuries after eruption of the Nevado del Ruiz volcano in Colombia in 1985, but gas gangrene equine antitoxin is of little use both because its efficacy is unknown and allergic reactions to it can be severe (M. Oxtoby, personal communication). Mass chemotherapy is not recommended, but in some cases chemoprophylaxis may be reasonable, such as for refugees newly arrived in an area with endemic malaria. Restraint in use of parenteral injections and appropriate care and use of injection equipment is indicated, as is screening of blood donors and donated blood.

Laboratory services are important but may be overused; not every person with a communicable disease needs to have laboratory confirmation of that fact. For communicable disease-control purposes, laboratory services are needed initially to determine the causative agent in representative cases so that appropriate control measures can be taken, and subsequently to document that the pathogen has been controlled. Laboratories that can do simple tests may be established in or near the disaster area; for more sophisticated tests, specimens need to be transported (in appropriate containers and conditions) to reference laboratories.

Surveillance. Perhaps the most important element of control of communicable disease after disasters is the establishment of effective surveillance (14,15). When there is no reliable information on the occurrence of infectious disease, rumors fill the void, panic may result, and political and public health leaders may be forced to waste resources on unwise and unneeded control measures. On the other hand, when the leaders are confident that they have current and reasonably comprehensive information on the occurrence of infectious disease, they can reassure the public with facts and can plan rational control measures as needed. The elements of an effective surveillance system for the purposes of communicable disease control include:

1. Identification of one person (preferably a national epidemiologist) whose primary responsibility is to maintain the surveillance system.

2. Concentration on diseases that are plausible consequences of the disaster or that are especially amenable to control.

3. Priority access to transportation to areas in which communicable disease problems are reported and to a reference laboratory so that appropriate specimens can be evaluated properly.

4. Prompt investigation of any unusual events detected by the surveillance system. Distinguishing disaster-related disease from the usual level of disease can be difficult because ascertainment of disease is often vastly improved after a disaster; any apparent increase may merely reflect better reporting.

5. Prompt investigation of any reports or rumors of outbreaks of communicable disease. Political sources, unofficial community sources, reports from relief workers, and newspaper accounts may well uncover disease problems that the established system has not identified. Scoffing at rumors without investigation may lead the media to suspect that they are observing incompetent bureaucracy at work, whereas sober investigation of rumors will instill confidence that the authorities are not trying to cover up problems instead of dealing with them.

6. Daily reporting to the central level even if no cases of communicable disease are seen, since central authorities may not be confident that no report means no disease. If diagnoses are unclear, symptom complexes may be reported in such terms as fever, fever and cough, fever and diarrhea, vomiting and/or diarrhea, and fever and rash. The reporting system should be kept simple and the number of reported diseases or symptom complexes kept small to gain and maintain the cooperation of the reporting units and to avoid overwhelming the various reporting levels with a large amount of data that must be analyzed promptly. A sample reporting form has been published (4).

7. Prompt analysis and dissemination of surveillance reports to all who are interested; secrecy will breed distrust and seriously detract from the value of the surveillance system. A sample form for a weekly summary of surveillance data at the central level has been published (4). Prompt analysis of the surveillance reports is crucial. Tabulation and analysis must not so overwhelm the epidemiologists that field investigations and disease-control activities are sacrificed. If this occurs, the process must be simplified or the tabulation delegated to other persons.

8. Continuation of the surveillance system until well after the emergency period, even though enthusiasm may wane rapidly. An outbreak of communicable disease may occur relatively late because the exposure was late, the incubation period was long (e.g., viral hepatitis), or the effect was delayed (e.g., an increase in the mosquito population).

Conclusion

Although outbreaks of communicable disease can occur after disasters, very few such outbreaks have been observed during the last 40 years (1,4,16). Although the lack of such outbreaks may partly reflect poor ascertainment and partly reflect effective countermeasures, the danger posed by communicable disease after a disaster is much less than is popularly perceived. Nevertheless, disasters carry elements that can contribute to transmission of disease, and persons responsible for managing disaster relief should establish a surveillance system and institute appropriate sanitary and medical measures.

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Earthquakes

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Introduction

An earthquake of great magnitude is one of the most destructive events in nature. In our everyday world in which the earth seems so unyielding, it is hard to imagine a force so great that it can shake the ground into standing waves several feet high; snap tree trunks in two; spill rivers and lakes over their banks; turn highways into strips of broken rubble; liquify alluvial soils and send spurts of sand and water into the air; tear open fissures in the earth more than 160 km (100 miles) long; generate seismic sea waves that can race across thousands of miles of open ocean at 700 km/hour (420 miles/hour) and still generate tidal waves up to 30 meters in height; send millions of cubic meters of rock, mud, and debris crashing down hillsides 160 km from the epicenter of the earthquake; and destroy virtually every structure in a city. Such force is hard to imagine, perhaps, on the scale of human endeavor, but easier to understand when viewed from a geologic perspective. The forces that generate great earthquakes are the same forces that generate continents, thrust ancient sea beds upward into great mountain ranges, and fold and break the earth's crust. When this enormous pent-up energy is suddenly released, the impact can mean disaster.

The United States has been relatively fortunate in terms of earthquake-related casualties—so far. On the scale of a human lifetime, earthquakes may seem somewhat random and unpredictable; however, on a geologic time scale, a major earthquake in a heavily populated urban area is inevitable. When a great earthquake eventually does strike one of our major urban centers, the destructive result is entirely predictable—a major catastrophe.

Factors Affecting Earthquake Occurrence and Damage

Natural factors

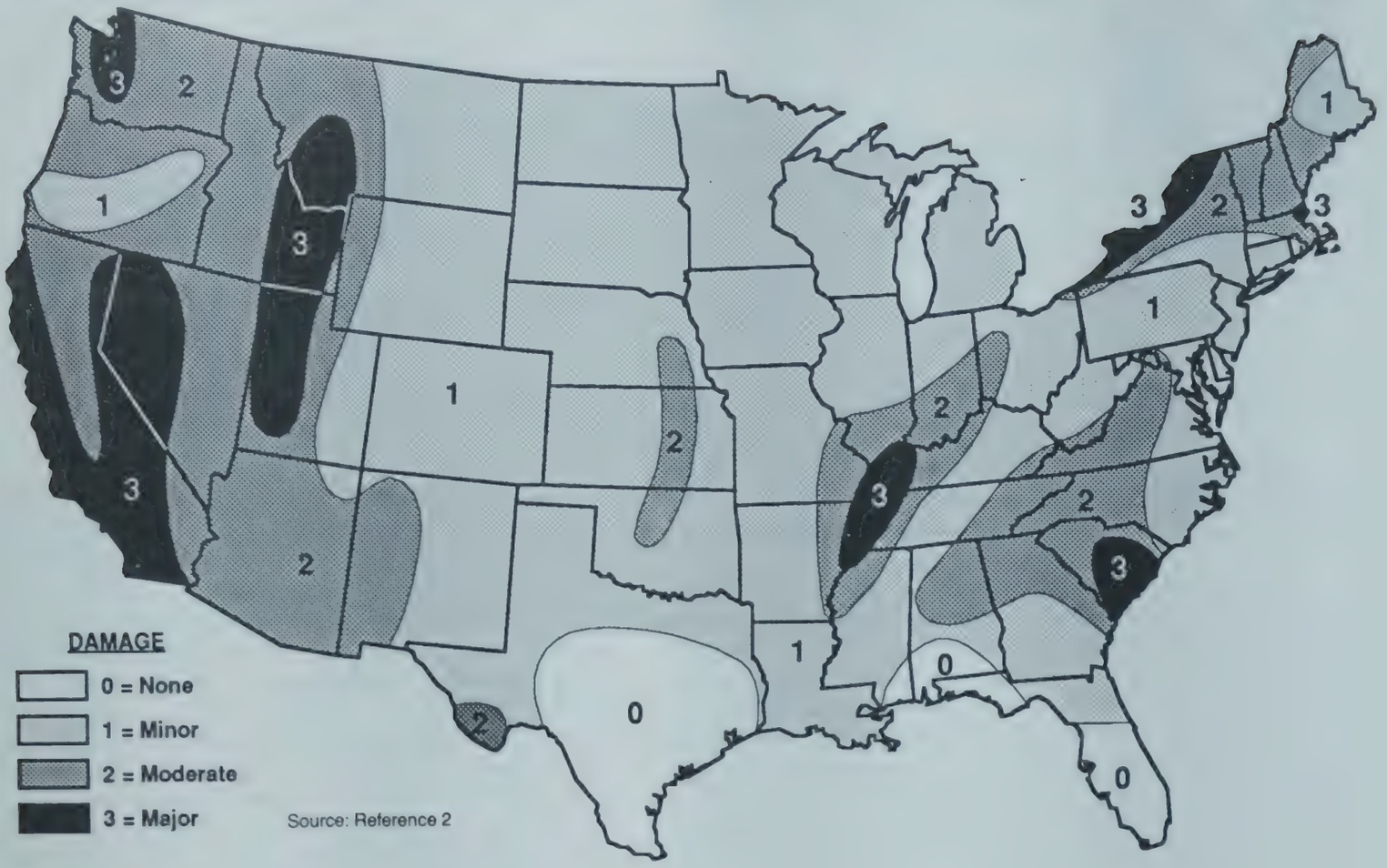
Geophysical factors. Plate tectonics, caused by the collision of the Pacific Plate with the North American Plate, generate most of the seismic disturbances along the Pacific West Coast and Alaska, particularly along the San Andreas Fault in California. Major fault lines exist where these two plates have collided. Forces build up as the two plates attempt to move with respect to one another, both laterally and vertically. Earthquakes occur when the adhesions along the fault give way, releasing pent-up energy. These large and inexorable forces are responsible for the band of seismic activity that extends along the Pacific rim to South America and to Japan. The rapid relative movement of these two plates contributes to the generally high rate of seismic activity. The relatively shallow depth of the San Andreas Fault (10-15 km below the earth's surface) adds to the perceived intensity of earthquakes along this fault.

Other areas of the United States have also been quite seismically active (Figure 1). Areas in which major earthquakes are probable include the Rocky Mountains, the upper Mississippi River and Ohio River valleys, South Carolina, and the area bounded by the eastern Great Lakes and New England. Alaska, Hawaii, Puerto Rico, and the Virgin Islands are all active seismic areas.In fact, throughout the United States, only a few Gulf Coast states are considered at very low risk.

Damaging earthquake forces are propagated as a series of compression waves (pushing and pulling in the direction of wave travel), shear waves (both side-to-side and vertical motion), and more complex waves with an elliptical or rolling motion. Waves travel outward from a fault at about 3-5 km/second, depending on the properties of the wave and the material through which it travels. Destructive ground shaking occurs at wave frequencies from about 0.1-30 Hertz (Hz) (cycles per second). High-frequency waves (>1 Hz) are more efficient at vibrating low buildings than are low-frequency waves, which are more likely to vibrate tall buildings. Low-frequency waves travel greater distances with less attenuation and can cause damage at great distances from the fault. Side-to-side motion is usually the most destructive because unreinforced buildings are less able to withstand it.

Depending on the properties of the earthquake and the local geology, natural and human-made structures are subjected to both vertical and horizontal wave motions that can set up destructive oscillations (swaying back and forth). A multistory building, for instance, acts as an inverted pen-

FIGURE 1. Seismic risk map for conterminous United States



dulum that oscillates at a certain frequency and amplitude (intensity) depending on its height, inertia, and structural characteristics. Seismic waves of the same frequency as the natural frequency of the building reinforce these oscillations and increase the damage. That the natural frequency of oscillation is a function of height explains why two buildings of similar construction but of different heights may experience substantially different degrees of damage.

Magnitude and intensity are two measures of the strength of an earthquake. Magnitude, the measure of physical energy released, is commonly calculated on the Richter scale, a logarithmic scale (to the base 10) of wave amplitudes (wave height as recorded on a seismometer, an earthquake-measuring instrument). On this scale, the recorded amplitude of a magnitude 8.0 earthquake is 10 times that of a 7.0 earthquake and 100 times that of a 6.0 earthquake. Recorded wave amplitude is related to the physical energy released in such a way that the energy released in a magnitude 8.0 earthquake is 31 times greater than the energy released in a magnitude 7.0 earthquake (1). Although the scale is open-ended, the strongest earthquake recorded to date has been of magnitude 8.9.

Intensity is measured by the perceived impact of the earthquake forces on the geologic strata through which the force is transmitted, as well as the direction and the amplitude of the seismic wave forces when they reach the surface. The most commonly used scale for intensity is the Modified Mercalli (MM) Intensity Scale (1), which ranges from barely perceptible earthquakes at MM I to near total destruction at MM XII. It is sobering to review the more intense categories on the Modified Mercalli scale:

CATEGORIES

V. Felt by nearly everyone; damage to contents and structures uncommon but possible.

VI. Felt by all; many frightened and run outdoors; damage slight.

VII. Everybody runs outdoors; damage negligible to buildings seismically well-designed and constructed; slight to moderate to ordinary structures; considerable damage to poorly built or badly designed structures.

VIII. Damage slight in well-designed, considerable in ordinary, and great in poorly built structures; chimneys, monuments, walls, etc., fall.

IX. Damage considerable to well-designed structures, and great (including partial or complete collapse) in other buildings; buildings shifted off foundations; underground pipelines disrupted.

X. Some well-built wooden structures destroyed; most masonry and ordinary structures destroyed; railroad tracks bent; landslides common; water spills over banks of streams, lakes, etc.

XI. Few, if any, masonry structures remain standing; bridges are destroyed; broad fissures open in the ground; underground pipelines are completely out of service; earth subsides.

XII. Damage is total; waves are seen propagating along surface of the ground; nearly impossible to stand; objects thrown up into the air.

The Richter and the MM scales do not correspond one-to-one. Two earthquakes that have the same given intensity on the MM scale may be several orders of magnitude apart on the Richter scale. Conversely, earthquakes of relatively modest magnitude may be quite intense in certain areas. In general, though, earthquakes of magnitude 6 correspond to MM intensities VII-VIII (moderate to major damage); magnitude 7, to MM IX-X (major damage the rule); and magnitude 8+, to MM XII (major to total damage certain).

The intensity of an earthquake is more germane than its magnitude to public health consequences. Intensity scales also allow comparisons with earthquakes that occurred before to the development of seismic monitoring instruments. The destruction that an earthquake causes is a function of its intensity and the resistance of structures to seismic damage.

Topographic factors. Topographic factors substantially affect the impact of earthquakes. The areas most susceptible are the outfall areas for landslides, mudslides, avalanches, and rock falls; low-lying areas susceptible to seismic sea waves (tsunamis or tidal waves) or floods from ruptured dams; and areas constructed on alluvial soils or landfill, both of which tend to liquify and exacerbate seismic oscillations.

Meteorologic factors. Meteorology plays only a minor direct role in the events that initiate earthquakes. However, it can substantially affect the secondary effects of earthquakes. High tides and high water levels from storm runoff exacerbate the impact of seismic sea waves. Water saturation of soils increases the likelihood of both landslides and avalanches and of earthen dams to fail, as well as increasing the probability of soil liquefaction during seismic shaking. Earthquake-induced failure of dams when streams are near flood stage would be catastrophic. If housing is substantially damaged, rain or subfreezing temperatures would be, at the least, a nuisance and could contribute substantially to morbidity and mortality.

Human-Made Factors

Structural factors. The single greatest risk for humans is from the collapse of structures. Human behavior has a substantial impact on how well designed and how carefully constructed these structures are, and, more importantly, whether they are built on or near seismically active faults. Ignorance, denial, expedience, and the all-too-familiar tendency to cut corners all weigh in the decision to locate structures in seismically unsafe areas and the failure to construct them using the proper aseismic techniques.

Technologic factors. The fire that followed the 1906 San Francisco earthquake caused more damage than the earthquake itself. Our modern industrial cities are laden with chemical and petroleum products that could contribute substantially to fire hazard as well as to the generation of toxic products of combustion and pyrolysis. In a major earthquake, underground pipelines carrying fuel, natural gas, and the city water supply can be expected to be disrupted. Immediate, widespread fires are also expected, along with impaired means to fight them. Industrial storage facilities for hazardous materials might leak and could cause widespread contamination of surface and groundwater, in addition to potential release of toxic vapors. Polychlorinated biphenyl-containing electrical equipment, if subjected to fire and pyrolysis, could cause substantial contamination by benzo-p-dioxins and dibenzofurans. Failure of a nuclear power plant resulting from seismic activity could lead to widespread contamination by radioactive materials.

Artificial causes of earthquakes. Three human activities have been known to induce earthquakes: a) filling large water impoundments, b) deep well injection, and c) underground explosions of nuclear devices. Some have speculated that nuclear detonations along a fault may release strain in a controlled fashion and prevent a major earthquake, but the potential liability of such an experiment gone awry has proved daunting for even the most intrepid seismic investigators.

Historical Overview of Major Damaging Earthquakes

Time Patterns

The science of predicting earthquakes is still in its infancy. Although some major earthquakes have been presaged by foreshocks, changes in groundwater and geothermal activity, and even animal behavior, most major earthquakes have occurred suddenly and without warning.

While the major earthquake event appears to occur somewhat randomly, periodicity has been observed along major faults. The two major San Andreas Fault zones in northern and southern California have had a major event on average every 140-150 years. Because it has now been 130 years since the magnitude 8.3 Ft. Tejon earthquake along the southern San Andreas Fault in 1857, the probability of a Ft. Tejon-sized event is thought to be greater than 1% per year, with a 40% risk over the next 30 years. In contrast, the northern San Andreas Fault, which had a major release in 1906, experienced no earthquakes greater than magnitude 5 until 1955. This latter part of the San Andreas fault has had an upsurge in seismic activity in the last few years, which some seismic experts believe increases the probability of a major earthquake. Activity along the fault will doubtless increase in frequency and severity until the next major event, with southern California the most likely site.

The south coast of Alaska and the Aleutian Islands is one of the most seismically active areas in the world, with nine earthquakes from 1938 to 1979 of magnitude 7.4 or greater. The 1964 Prince William Sound earthquake of magnitude 8.3 is the second strongest earthquake of the 20th century and one of the most violent earthquakes ever recorded, releasing seismic energy equivalent to the current total annual energy consumption in the United States. Several areas of apparent unrelieved strain along the tectonic plate boundary are likely sites of future major earthquakes. The repeat intervals along the plate boundary average 50-100 years.

The eastern United States has considerably less activity than the area west of the Rocky Mountains, but a damaging earthquake occurs about every 25 years and a great earthquake, every 50-100 years. A series of three great earthquakes (estimated magnitude 8.6, 8.4, and 8.7) all of intensity XII occurred over a 3-month period in 1811-1812 near New

Madrid, Missouri. Although little loss of life occurred in the then sparsely populated area, the earthquakes were felt over most of the United States east of the Rocky Mountains and caused destruction for hundreds of miles. The New Madrid fault system is less well studied than the San Andreas system, but New Madrid-sized earthquakes may recur at 600- to 700-year intervals. Enough strain may have developed to produce a magnitude 7.6 earthquake, which would be damaging over 200,000 square miles. Charleston, South Carolina, experienced a magnitude 6.8 (intensity X) earthquake in 1886 that killed 83 people and was felt over most of the United States east of the Mississippi River. The specific fault responsible has not been identified, but similar geologic conditions exist over much of the mid-Atlantic seaboard with uncertain probabilities of damaging earthquakes. The lower probability of major earthquakes in the East needs to be balanced against the greater population densities, less stringent seismic codes, and the fact that earthquakes in the East exert damaging effects over a much wider area for an event of a given magnitude.

Trends over Time

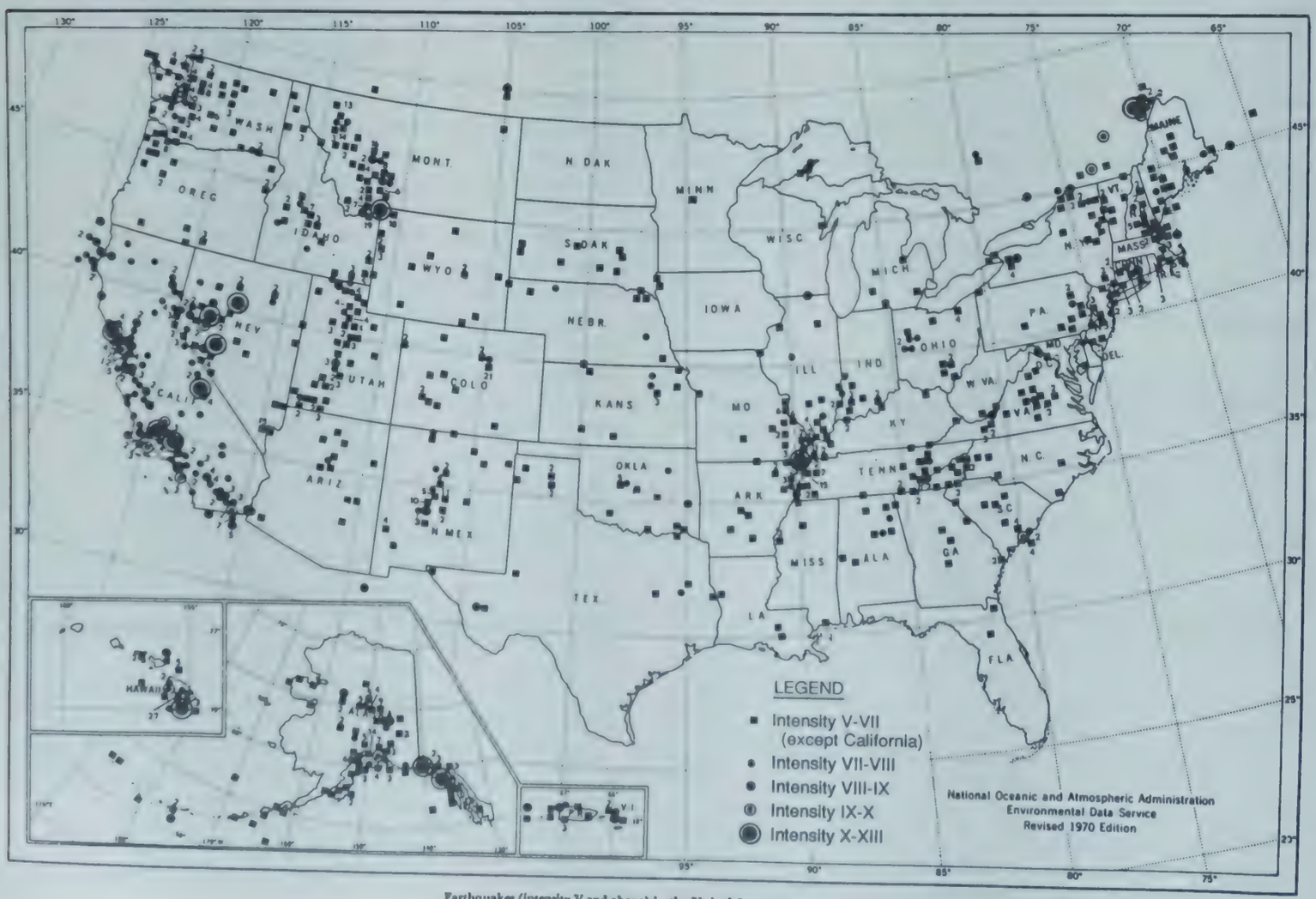
At present we have only a rough idea of the probability of a major earthquake's occurring in a given area during a given interval of time. There is no reason to believe that earthquakes are either increasing or decreasing in frequency or intensity. However, the population at risk has dramatically increased in seismically active areas. Aside from restricting population growth in these areas (a measure usually ignored), the only practical way to control earthquake hazards is to develop and enforce effective seismic safety codes and to phase out older structures that do not meet the codes.

Geographic Areas at Risk

Earthquakes occur on a geologic time scale as a result of forces set in motion millions of years ago. Current areas of high seismic activity will continue to be at high risk for the foreseeable future. The earthquake history of the United States has been described in some detail in a compendium (2). The compendium's graphic descriptions of the effects of a major earthquake are riveting. With the exception of colonial records in the Northeast that date back to 1638, the historical record of U.S. earthquakes is scarcely 2 centuries old, a very small period of observation on the geologic time scale. The zones of various levels of seismic risk are shown in Figure 1. The locations of the epicenters of significant earthquakes through 1970 are shown in Figure 2 and for the years 1971-1980 in Figures 3a and 3b. Approximately 90% of the seismic activity in the contiguous United States occurs in California and western Nevada.

Earthquakes often occur in association with active volcanoes, sometimes triggered by magmatic flow and sometimes releasing pressure that allows magmatic intru-

FIGURE 2. Epicenters of significant earthquakes, United States, 1970



Earthquakes (intensity V and above) in the United States through 1970.

FIGURE 3A. Epicenters of significant earthquakes, conterminous United States, 1971-1980

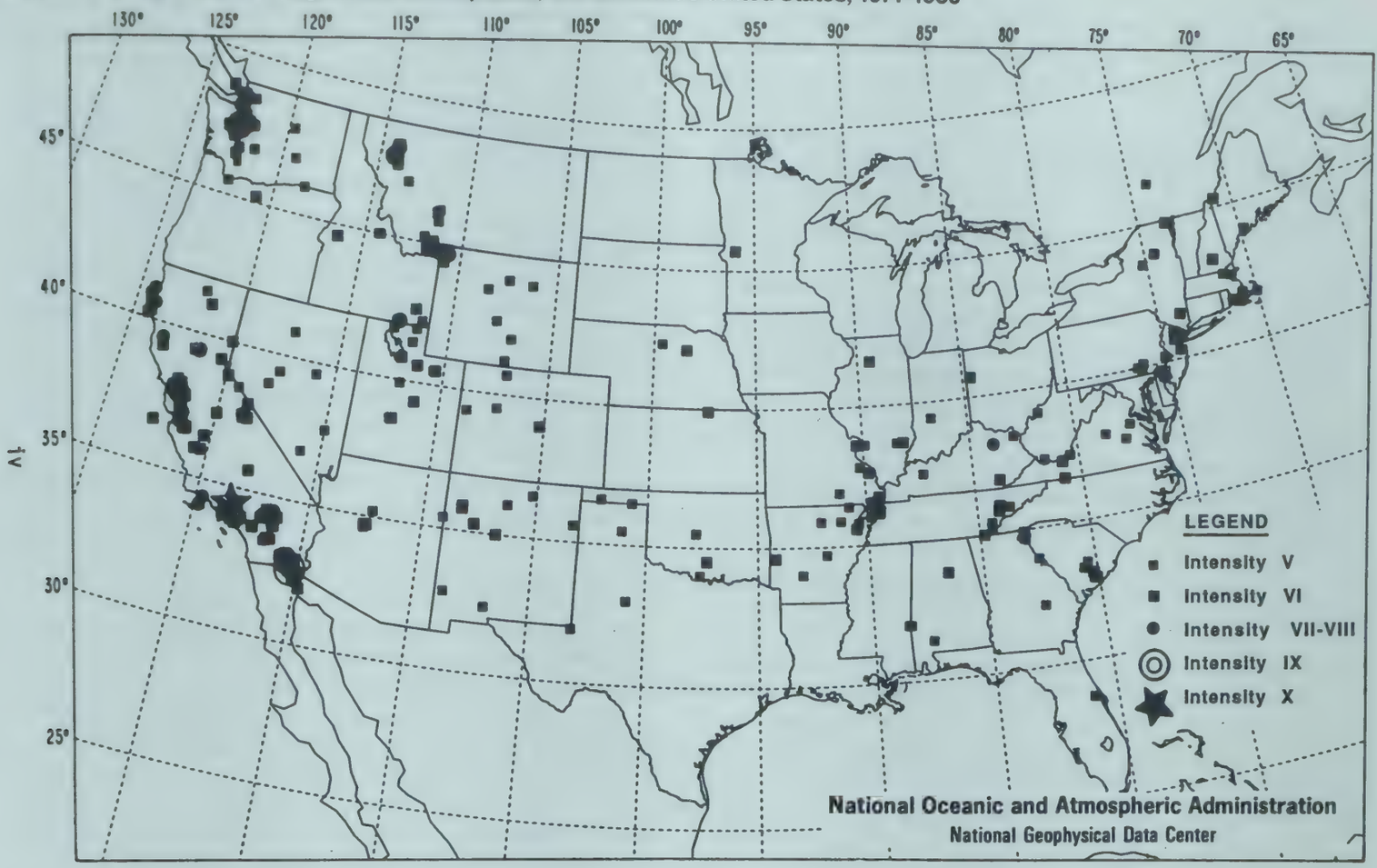
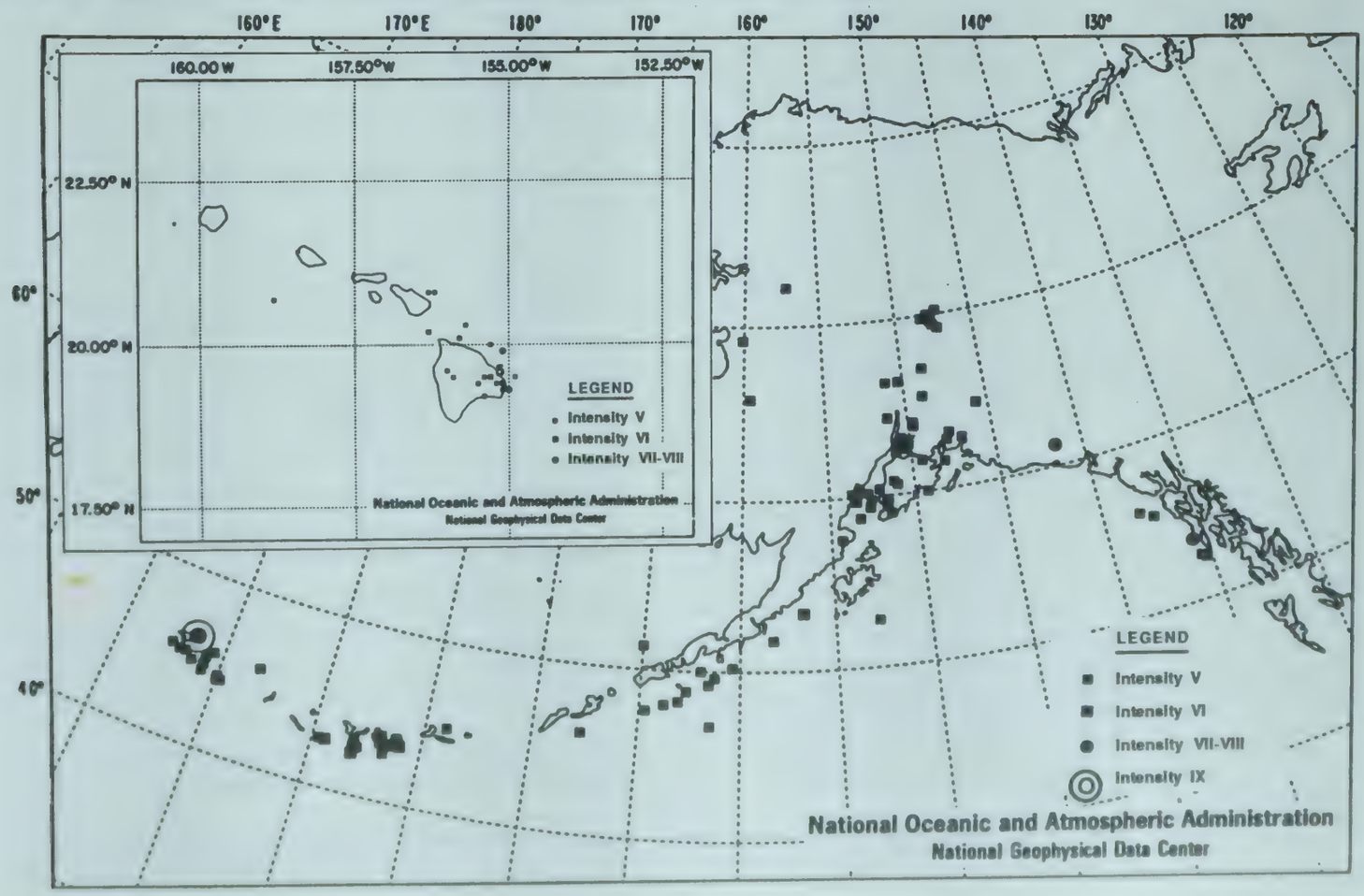


FIGURE 3B. Epicenters of significant earthquakes, Alaska and Hawaii, 1971-1980



sion. The so-called harmonic tremors associated with actual magmatic flow are generally not damaging. Relatively severe earthquakes can immediately precede or accompany volcanic eruption and can contribute to devastating mudslides.

Earthquake Morbidity and Mortality Risk Factors

Approximately 1,600 deaths attributed to earthquakes have been recorded in the United States since colonial times. More than 1,000 of these deaths have occurred in California, including 700 in the 1906 San Francisco earthquake (Table 1). As a cause of death, hurricanes, floods, and tornadoes have each exceeded this relatively modest total. However, there is little cause for complacency. Population growth in areas of high seismic risk has greatly increased the number of people at risk since the last earthquake of great magnitude struck. Populated areas in other parts of the world have suffered great loss of life from catastrophic earthquakes, e.g., Guatemala 1976 (22,778), Peru 1970 (52,000), Japan 1923 (142,807), and China 1976 (665,000). A single great earthquake in one of our cities could result in exceeding the death toll associated with all the natural disasters ever experienced in the United States.

In contrast to the rich and prolific literature on earthquake engineering and earthquake geology, little detailed epidemiologic information on earthquake morbidity and mortality is found in the scientific literature. Most reports are limited to fairly general summary statistics on deaths and injuries or to anecdotal descriptions of disaster medical relief, but some laudable exceptions exist. While we can speak relatively confidently of the major hazards associated with earthquakes, few earthquakes have been the subject of detailed epidemiologic study.

Trauma caused by partial or complete collapse of human-made structures is the overwhelming cause of death and

TABLE 1. Selected fatal earthquakes in the United States, 1811-1987

Date	Location	Magnitude	Deaths	Dollor loss (in millions)
16 Dec 1811	New Madrid, Mo.	8.6	few	NR*
23 Jan 1812	New Madrid, Mo.	8.4	few	NR
07 Feb 1812	New Madrid, Mo.	8.7	few	NR
08 Dec 1812	San Juan Capistrano, Calif.	6.8	40	NR
09 Jan 1857	Ft. Tejon, Calif.	7.9	1	NR
21 Oct 1868	Hayward, Calif.	6.8	6	NR
26 Mar 1872	Owens Valley, Calif.	7.8	27	NR
31 Aug 1886	Charleston, S.C.	6.8	83	NR
19 Apr 1892	Vacaville, Calif.	6.8	1	NR
25 Dec 1899	San Jacinto & Hemet, Calif.	6.7	6	NR
18 Apr 1906	San Francisco, Calif.	8.3	700	500
22 Jun 1915	El Centro, Calif.	6.3	6	1
21 Apr 1918	San Jacinto & Hemet, Calif.	6.8	1	NR
11 Oct 1918	Mona Passage, P.R.	7.5	116	29
29 Jun 1925	Santa Barbara, Calif.	6.3	13	8
29 Jun 1926	Santa Barbara, Calif.	5.5	1	NR
06 Jun 1932	Humboldt County, Calif.	6.4	1	NR
11 Mar 1933	Long Beach, Calif.	6.3	15	40
19 Oct 1935	Helena, Mont.	6.2	2	19
31 Oct 1935	Helena, Mont.	6.0	2	6
19 May 1940	Imperial Valley, Calif.	6.7	9	33
01 Apr 1946	Unimak Island, Alaska	7.4	173	90
13 Apr 1949	Olympia, Wash.	7.0	8	80
25 Jul 1952	Kern County, Calif.	7.7	12	150
22 Aug 1952	Bakersfield, Calif.	5.8	2	30
21 Dec 1954	Eureka, Calif.	6.6	1	6
23 Oct 1955	Walnut Creek, Calif.	5.4	1	3
09 Jul 1958	Lituya Bay, Alaska	7.9	5	NR
18 Aug 1959	Hebgen Lake, Mont.	7.1	28	26
27 Mar 1964	Prince William Sound, Alaska	8.4	131	1,020
29 Apr 1965	Seattle, Wash.	6.5	7	28
09 Feb 1971	San Fernando, Calif.	6.8	65	900
29 Nov 1975	Kalapana, Hawaii	7.2	2	5
01 Oct 1987	Whittier, Calif.	5.9	9	350

*Not reported

Source: Data compiled from references 2, 20, 21, 22, and 23. Some dollar amounts are historical figures, while others were adjusted to 1979 dollars by the authors of reference 20.

injury in most earthquakes. Rockslides, snow avalanches, and landslides are important threats in hilly and mountainous areas. Tsunamis can cause great loss of life by inundating low-lying coastal areas. Transportation casualties can occur from train derailments, collapse of bridges and highway overpasses, and capsizing of boats by seismic sea waves. Indirect hazards include post-earthquake fires, hazardous chemical and radiation release, electrocution, injury during rescue or clean-up operations, acute myocardial infarction and exacerbation of other chronic diseases, anxiety and other mental health problems, respiratory disease from exposure to dust and asbestos fibers from rubble, and flooding from the broken dams. Infectious epidemics after earthquakes have generally been conspicuous by their absence, despite popular concern. Some of these earthquake-related hazards are worth exploring in more detail.

Direct Hazards

RISK FACTORS RELATED TO STRUCTURAL COLLAPSE

Poor design and materials. Unreinforced masonry (including adobe) is the most hazardous building material and can be expected to fail in even moderate earthquakes. In addition to being structurally weak, masonry is doubly hazardous because of its weight and potential for causing injury when falling. Catastrophic failure of "modern," medium-rise, concrete-slab buildings from collapse of their supports has been well described, e.g., the 1964 Anchorage (3) and 1971 San Fernando (4) earthquakes, and played a prominent role in damage associated with the 1985 Mexico City earthquake.

Buildings can be designed to various levels of seismic security. At a minimum, they should be designed so that the occupants can survive, even if the building is irreparably damaged. At the next level of design, the building remains functional even though damaged (an important design criterion for hospitals and other public works that must be able to operate after an earthquake). Structures meeting the most stringent level of design will withstand an earthquake with little or no damage.

Unfortunately, most of our older buildings and many of our newer ones fail to meet even the minimum standard for earthquake-proofing. The principal design flaw in most construction is its inability to withstand lateral forces, usually compounded by weak materials that fail under the seismic load. Although there has been much progress in engineering design, seismic safety is still an inexact science. The Olive View Medical Center, newly constructed to seismic codes, was destroyed by the 1971 San Fernando earthquake 1 month after its dedication (5)

Poor construction. Good design required by seismic codes can be negated if builders cut corners on materials and construction technique. Rigorous enforcement of building codes can prevent shoddy and below-code-level work.

Falling objects. Heavy furniture, appliances, bookshelves, equipment, and objects placed high can fall and cause injury unless secured. Because chimneys are particularly vulnerable to failure, persons exiting buildings during seismic shaking should avoid the potential path of falling brick.

Dust from rubble. Heavy dust has been reported immediately after earthquakes and for a considerable time afterward (6). For trapped victims, heavy dust can be a life-threatening hazard (7). Dust has hampered rescue and clean-up operations by causing eye and respiratory-tract irritation. Anecdotal accounts from the 1985 Mexico City earthquake report that rescue workers finally resorted to full-face respirators, equipment that will probably be in short supply after a major earthquake. Commercial and school construction in the United States is often heavily laden with asbestos, which will likely pulverize if subjected to collapse. The asbestos and other particulate matter in the dust could pose both subacute and chronic respiratory hazards to rescue and clean-up personnel, depending on the characteristics and toxicity of the dust.

RISK FACTORS FOR OCCUPANTS

A study of housing-related earthquake injuries in a Guatemalan village of principally adobe construction showed that the young and the elderly were at highest risk of injury and death (8). The youngest child, who usually slept with the mother, tended to share the more favorable mortality risk of the parents. Women of all ages were found to have a higher rate of serious injury. Other than age of the adobe bricks, no structural features seemed to relate to injury rates in adobe housing. Occupants of non-adobe housing fared much better. Large family size was found to relate to increased rate of injury.

An ambitious study conducted after the 1980 earthquake of 3,619 villagers in southern Italy, in contrast to the Guatemalan study, found no differences in injury or death rates by age or sex (9). Despite these well-documented findings in Italy, the consensus in the literature is that the very young, the elderly, and the chronically ill are at somewhat higher risk of death or injury in earthquake disasters. Lack of mobility to flee collapsing structures, inability to withstand trauma, and exacerbation of underlying disease are all plausible reasons for believing that these groups are more vulnerable.

In the Italian study, entrapment requiring assistance to escape was the most important risk factor (death rate was 35.0% for trapped versus 0.3% for untrapped persons), but entrapment was also strongly correlated with the degree of structural damage. Interestingly, death and injury rates were similar for those inside the home and those outdoors at the time of the quake, but were twice as high for those in bars and dancing places. Occupants of upper floors fared less well than ground-floor occupants. Those who ran from the building during the quake fared better than those who did not.

A number of important observations were made about the source, timing, and outcome of injuries. All the deaths and injuries that occurred in the first 48 hours after the earthquake were due to structural collapse. Virtually all injuries (97%) occurred immediately or within 30 minutes of the quake. Survival among the trapped fell off rapidly with time, with 88% alive Day 1, 35% alive Day 2, 9% alive Day 3, and no survivors among 77 bodies extricated on Day 4 on (10). Of all the trapped who were extricated alive, 333 (94%) were rescued during the first 24 hours. People living alone tended to be extricated later, possibly because they did not have family members to rescue them; they also had a higher fatality rate. However, their homes tended to be more heavily damaged. Nearly all the people who died (95%) were trapped and died before rescue. The probability of survival after

being freed was high. After the first 48 hours the survival rate for the injured was the same as for the uninjured. Investigators concluded that substantial reduction in mortality could be attained only by improving rescue rates within the first 48 hours.

Behavior of occupants. Does standing in a doorway or crawling under a desk really improve chances of survival? Can people think to take evasive action when their world is disintegrating around them and they have but a few seconds to react?

Anecdotal reports suggest that some people may be too stunned or frightened to think rationally or even to move (11). Other anecdotal reports suggest the efficacy of moving to a protected area such as a doorway or under a desk, but behavior of occupants during and immediately after an earthquake has been poorly studied. From the 1985 Mexico City earthquake, anecdotal reports of little islands of concrete slab perched on the tops of childrens' school desks while the rest of the ceiling had collapsed to the floor suggest that earthquake drills might be worthwhile. The real question, of course, is whether the children would have been able to get under the desks in time to prevent injury if the school had been occupied.

Earthquakes, although sudden, are usually not instantaneous. There are often a few seconds to react before the shaking reaches maximum intensity, raising the possibility of taking evasive action to escape injury. In the best-documented study report identified, 118 employees of a county office building in Imperial County, California, were studied after a magnitude 6.5 earthquake damaged their building (12). The period of strong shaking lasted 8 seconds. The investigators had an unusual opportunity to study behavior of occupants because everyone survived and because completing the questionnaire was required by all employees. Of the 118 office workers, 37% got under a desk, 15% stood in a doorway, 37% stayed where they were, 3% went into a main corridor, 2% left the building, 8% dodged falling objects, and 14% did something else. Of interest is that 30% of the desks under which people sought refuge moved away during the shaking. The occupants attributed their actions as follows: 18% to previous drills in elementary school, 27% to fire and bomb drills in the building, and 25% to experience with previous earthquakes (multiple responses permitted). Because no major building components collapsed onto the workers, the efficacy of their actions is hard to judge.

Injury of occupants. An extensive earthquake planning scenario has been developed for a magnitude 8.3 earthquake along the San Andreas Fault (13). The emergency-medical-services part of the scenario anticipates up to 25,000 deaths and up to 100,000 seriously injured (14). Most planning scenarios call for a ratio of 3.0-3.5 injuries/fatality, but victims trapped in collapsed structures may have a fatality rate high enough to decrease the ratio of injured survivors. The problems anticipated for an urban industrial area obviously involve more than the straightforward lacerations (52%), contusions (27%), and fractures (19%) caused by structural collapse in the Italian villages (9). The conditions in the southern California scenario are anticipated to be 27% surgical, 23% orthopedic injuries, 15% major medical/cardiac, 10% neurosurgical, 10% shock (as the primary problem), 6% severe burns, 5% smoke and toxins, and 5% psychiatric.

Chronic sequelae of neurologic injury, especially spinal-cord injuries, can be expected. A rate of 1.5 cases of paraplegia/1,000 injured was observed after the Guatemalan earthquake (15). Amputations and other chronic orthopedic conditions can be expected.

After a magnitude 6.7 earthquake in Athens, Greece, a 50% increase in cardiac deaths was observed for the first 3 days, peaking on the third day (16). The authors attributed the increase to psychological stress because no increases in other causes of death (including trauma) were seen. This study of death certificates, however, did not allow the examination of alternative hypotheses such as the effects of exertion from clean-up activities.

ROCKSLIDES, SNOW AVALANCHES, AND LANDSLIDES

It is not practical to try to construct buildings that will withstand a landslide or similar insult. The only reasonable alternative is to exercise due care and discretion in locating structures well away from potential areas of impact. One interesting report from a magnitude 7.2 earthquake on Guadalcanal in 1977 suggests that people might escape slides by running uphill where the apex of the slide is narrower (17). Although there were no housing-related deaths (due to the light construction of housing), there were 12 deaths from landslides, primarily in the terraced garden areas of logged hillsides. Landslide was the dominant feature in an earthquake in the Kansu Province of China that killed 100,000 in 1920 (18).

TSUNAMIS

Low-lying areas along seacoasts and around bays and harbors are at risk of inundation by seismic sea waves, which the Japanese call tsunamis (for "harbor wave"). A tsunami can be directly propagated by crustal motion during earthquakes or by landslides, including underwater landslides. Tsunamis can travel enormous distances at 300-600 mph with very little loss of energy. Wave heights in deep ocean water may be only a few feet and pass under ships with little disturbance, but in shallow coastal waters wave heights can reach 100 feet with devastating impact on local shipping and shoreline areas. Successive crests may arrive at intervals of 10-45 minutes for several hours.

The Pacific Coast is at greatest risk from tsunamis, primarily from earthquakes in South America and the Alaska/Aleutian Island region. The 1964 Alaska earthquake generated tsunamis up to 20 feet in height along the coasts of Washington, Oregon, and California and caused extensive damage in Alaska and Hawaii. The death toll from these tsunamis was 122 compared with nine from the earthquake itself. Tsunamis are the leading earthquake-related problem in Hawaii.

The Japanese have historical accounts of tsunamis dating back to A.D. 684 and have constructed breakwaters and other counter-tsunami measures (19). It would be prudent to place no residential construction in tsunami-impacted areas and only necessary commercial structures at the shoreline, e.g., docking facilities and wharfs. Prompt evacuation of low-lying areas should be a priority disaster-response effort when a warning is issued by the National Oceanic and Atmospheric Administration's tsunami warning network headquartered in Hawaii. Tsunamis generated by nearby earthquakes, however, may give little or no war-

ning, making mortality a function of population density in the low-lying areas.

Public Health Implications of Earthquakes

Prevention and Control Measures

Primary prevention of earthquakes is obviously impossible, but much can be done to prevent the adverse consequences of an earthquake. Given the usual longevity of our buildings, most buildings in the United States, including virtually all construction on the West Coast, can be expected to be subjected to a least one episode of strong shaking. The major determinants of death and injury will already be in place at the moment the earthquake strikes. Disaster response in the aftermath of an earthquake, though important, can affect only a fraction of the adverse outcomes.

The challenge for our society is that more than 90% of what needs to be done must have been done well in advance of the earthquake and involves a conscientious investment in seismic safety year after year, often without interim earthquakes locally to remind us of the necessity to maintain the effort. With great earthquakes occurring in any one area on geologic cycles of 50-150 years or more, it is essential to develop the long view toward seismic safety. However, adopting and maintaining the long view is not an approach for which our local planning commissions and our democratic processes are noted. Public health and disaster-response officials need to join in the effort to develop and maintain an effective seismic safety-planning and enforcement program.

PRIMARY PREVENTION OF EARTHQUAKES

Although we can neither prevent earthquakes nor set off small ones to prevent big ones, we should take earthquakes into consideration before undertaking activities known to precipitate earthquakes, such as making deep well injections, filling water impoundments, and discharging underground nuclear explosives.

AVOIDANCE OF CONSTRUCTION IN AREAS OF HIGH SEISMIC RISK

Avoiding unnecessary residential and commercial construction on or near active faults and in areas subject to tsunamis, slides, and soil liquefaction is technically a secondary prevention measure for earthquakes, but a primary prevention measure for earthquake-related injuries. Areas of high seismic risk have been fairly well delineated, and the information should be available to local planners and developers. From a public health point of view, it is preferable to avoid construction in areas of high risk rather than to rely on seismic construction design to withstand the hazard, particularly since there have been some spectacular failures of supposedly "earthquake-proof" design.

In the last 20 years in San Francisco, for example, a veritable forest of high-rise buildings has been constructed, and there is considerable commercial pressure to extend the downtown area onto areas of landfill and bayfill. Despite the fact that these designs have not been subjected to the ultimate empirical test of a strong earthquake, the existence of the current buildings is used to argue that further construction is justified. Major metropolitan areas can be and have been built in the intervals between great earthquakes, especially in California. Due caution in site selection is in order for what is, in essence, an experiment in seismic design and construction on a grand scale.

DEVELOPMENT AND ENFORCEMENT OF SEISMIC SAFETY CODES

Land-use codes and building construction codes appropriate to the level of seismic risk should be developed and rigorously enforced. Aseismic design is an evolving science, and codes need to be updated periodically to reflect what has been learned from building performance during actual earthquakes. Particular attention should be paid to areas in the East and in the upper Mississippi River valley, where actual risk may be higher than perceived and where, consequently, local codes may not be adequate. How and when and at what expense older buildings should be brought up to code is a major public health issue since these buildings are likely to be the most vulnerable. Because structural collapse is the single greatest threat to life, seismic construction should be the number-one earthquake priority for most communities. In addition to the attention given homes, schools, and office buildings, due care should be given to industrial facilities with flammable, explosive, or toxic threats and so-called lifeline facilities such as hospitals, power plants, municipal waterworks, and transportation centers. Although often beyond the purview of building codes (or any reasonable hope of enforcement), heavy furniture, appliances, and objects placed where they could fall or be thrown about should be secured to prevent them from striking people in the event of an earthquake.

PREDICTION OF EARTHQUAKES

If earthquakes and damaging aftershocks could be predicted reliably, many lives could be saved by evacuating unsafe structures. Many earthquake predictions must necessarily be couched in terms of days or weeks or longer. It is doubtful that evacuations could be maintained for any but the most imminent of predictions, and even then, not for very long. Tsunami prediction, although better developed than earthquake prediction, can sometimes result in false alarms. More disturbing, however, is that many people react to tsunami warnings by going to the waterfront to "watch the waves come in," ignorant of the true potential for disaster.

DRILLS FOR EVASIVE ACTIONS DURING EARTHQUAKES

Despite the relative lack of data on efficacy of occupant behavior, it seems worthwhile for individuals to practice taking evasive actions (such as standing in a doorway, crawling under a desk, or quickly running outside), particularly since they will have just a few seconds to act when an earthquake strikes.

PLANNING SCENARIOS FOR EARTHQUAKES

Relative chaos is likely to prevail immediately after a major earthquake. Area residents, cut off from the outside, will initially have to rely on self-help. They can best help themselves and others if they have already worked out the scenario and practiced the necessary skills. First-aid skills should be taught. How medical triage facilities will be set up and tied in with the existing hospital and emergency medical

services networks should be decided. Strategies for rescuing trapped victims with available equipment should be developed. Response scenarios for toxic hazards should be thought out in advance, because specialized technical expertise is unlikely to be available to responders after an earthquake.

DISASTER RESPONSE TO EARTHQUAKES

Disaster response to earthquakes is more akin to medical treatment than to prevention, but some aspects of the response may be likened to tertiary prevention in that they seek to stem further injury and to control the secondary effects of the earthquake. Prompt rescue should improve outcome. Early treatment of injured persons should lessen sequelae. Provision of adequate food, water, and shelter should especially help persons in vulnerable age groups and those with pre-existing disease. Effective environmental-control measures should prevent secondary environmental health problems. Identification and control of long-term hazards (e.g., asbestos in rubble) should reduce chronic effects.

Surveillance

EARLY RAPID ASSESSMENT OF IMPACT

Because rapid rescue of trapped victims and prompt treatment of persons with life-threatening injuries can improve outcome, early rapid assessment of the extent of damage and injuries is needed to help mobilize resources and direct them as they are most needed. Unfortunately, the very factors likely to cause large numbers of injuries are also likely to disrupt communications and transportation and to damage medical-care facilities. Public health officials need to establish in advance how the impacted areas will be surveyed. For instance, a helicopter fly-over to assess damage according to a predetermined sampling grid (rather than a nonsystematic survey or anecdotal field reports) would likely yield a more accurate estimate of damage and potential injuries.

SURVEILLANCE OF INJURIES AT MEDICAL TRIAGE CENTERS

Triage centers should designate someone to organize surveillance of injuries, collect data, and see that the data are tabulated and reported to disaster-response health officials. In addition to collecting adequate information on the location and severity of the injury and disposition of each patient, the surveillance team should attempt to record a permanent point of contact for individuals outside the disaster impact area so that follow-up studies and/or surveillance efforts can find them, even if earthquake damage results in their not returning to their previous addresses. Depending on the urgency of the situation, some information can be collected on the spot about how an injury was sustained. Collecting good basic data at the outset will both provide decision makers with accurate data on injuries and form the basis for learning lessons applicable to the next earthquake.

SURVEILLANCE OF SEARCH AND RESCUE

Much can be learned from the position and circumstances of trapped victims, including persons who have died. Ideally, search and rescue teams should have surveillance forms to record important information about building type, address, nature of collapse, degree of dust, fire or toxic hazards, location of victims, nature and severity of injuries and other factors. Victims pronounced dead at the scene should be tagged with an identification number so that the medical examiner's data can later be linked to the search and rescue surveillance form. Surveillance of search and rescue activities should be used to direct resources to sites where the most good can be done in the first 24-48 hours, the most critical time.

SURVEILLANCE OF DISEASE

Rumors and fears of epidemics generally circulate in the aftermath of disasters, and earthquakes are no exception. Health officials should be prepared to recommend appropriate sanitary precautions and to dispel unfounded rumors and inaccurate information. They should set up a disease surveillance mechanism appropriate to the circumstances and provide regular reports to disaster-response officials. Unusually high incidence of disease should be investigated and control measures implemented. Outbreaks of infectious disease generally have not followed earthquakes in other countries and are unlikely to occur in the United States.

DETAILED FOLLOW-UP EPIDEMIOLOGY

Few earthquakes have been adequately studied epidemiologically, with the exceptions previously noted. It is vital that plans for follow-up epidemiology be developed before an earthquake occurs so that initial surveillance data collected will allow proper follow-up. Disaster-response officials need to be convinced to invest time and resources in the initial surveillance effort, even though their attention is likely to be focused on emergency medical services and disaster relief. Without this investment, the opportunity to learn many lessons useful for future earthquakes may be lost. Again, it is important to take the long view toward the certainty that earthquakes will recur and set aside the resources to study these natural experiments when they occur.

DISSEMINATION OF INFORMATION

Telephone service is likely to be disrupted in the impact area of an earthquake. Police, fire, and many emergency-service organizations maintain radio networks, which public health officials will need to use. Radio and television news crews often arrive at the scene of a disaster with sophisticated communications equipment. Public health organizations should work out scenarios for various information-dissemination contingencies before an earthquake occurs. The electronic news media can be an effective vehicle for health advisories, tsunami warnings, and updates on casualties and relief efforts. Ideally, public officials should work out media guidelines for information dissemination so that all parties will know what to expect when the disaster strikes. Epidemiologists should be aware that computer modems allow radio transmission of digital data. With the appropriate power supplies and information-dissemination devices, data from triage centers might be sent to a central collection point, facilitating surveillance.

Research Needs

Many of the deficiencies in our earthquake public health database have been noted. The most pressing research needs from a public health point of view involve the following areas:

1. Prediction of earthquakes and damaging aftershocks.

2. Prediction and appropriate hazard warnings for tsunamis.

3. Aseismic construction techniques, especially retrofitting of older buildings.

4. Epidemiology of earthquake-related injuries, including whether occupant behavior makes a difference in avoiding injuries.

5. Improved techniques for locating and rescuing trapped victims rapidly.

6. Rapid assessment techniques immediately after earthquakes to allow appropriate allocation of relief efforts.

7. Improved assessment of the potential threat of toxic releases and other nontraditional earthquake hazards that are a function of our modern industrial cities.

8. Improved assessment of the hazard represented by dust and appropriate control measures.

Summary

A major earthquake in one of our urban areas ranks as the largest potential natural disaster for the United States. Most of what can be done to mitigate injuries must have been done before the earthquake occurs. Because structural collapse is the single greatest hazard, priority should be given to seismic safety in land-use planning and in the design and construction of buildings. We need to improve prediction methods for earthquakes and tsunamis. Earthquake-disaster-response planning should concentrate on the rescue and care of trapped or injured victims as quickly as possible. Careful attention should be paid to accurate surveillance and the epidemiologic follow-up of earthquake disasters. Because of the relatively long time between major earthquakes, the public health community faces a special challenge in effectively communicating the hazard and the necessity to plan and take action before an earthquake occurs. It is inevitable that a major earthquake will strike one of our cities again, and we must be prepared.

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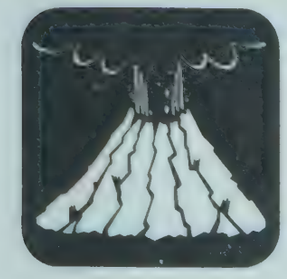

Volcanoes

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Background and Nature of Volcanoes

The eruption of Mount St. Helens in 1980 was the first volcanic disaster to occur in the United States in recent times. Apart from volcanoes in Hawaii, to be mentioned later, the most recent previous eruptions were a minor one at Mount Lassen, California, in 1914 and the massive eruption of Mount Katmai in 1912, a then-remote Alaskan peak. Since 1980, explosive eruptions have occurred around the world in populated areas such as at Pagan, Mariana Islands; El Chichon, Mexico; Galunggung, Indonesia; Mayon, Philippines; and, catastrophically, at Ruiz, Colombia, where in 1985 over 23,000 people died in a vast mud flow.

Although the Mount St. Helens eruptions in 1980 led to the relatively small number of 67 deaths, the scale of the May 18th event and the widespread ashfalls reawakened interest in volcanic hazards and provided the impetus for numerous scientific investigations, particularly in the area of public health. It is scarcely an exaggeration to say that before 1980 medical information on health hazards associated with volcanic activity was largely restricted to a few anecdotal or sparse accounts.

Distribution and Types of Volcanoes

About 500 volcanoes worldwide were active in historic times, and 100 or more of these have been notorious for the frequency or severity of their eruptions in populated areas (1,2). Some notable eruptions are listed in Table 1 (3). Most volcanoes, including those in the United States, are in a belt bordering the Pacific Ocean and referred to as the "Ring of Fire." A second belt stretches from southeastern Europe through the Mediterranean and southern Asia into Indonesia, where historically most deaths from volcanic eruptions have occurred. In addition, isolated volcanoes are found in the Pacific, Atlantic, and Indian Oceans. Most volcanoes lie within 300 kilometers (186 miles) of the sea in earthquake zones.

About 400 of the 500 known active volcanoes in the world lie where tectonic plates undergo compression (subduction zones), e.g., the Pacific "Ring of Fire," and these tend to be explosive. Explosive volcanoes can erupt violently and release large quantities of ash. Volcanoes in Hawaii and Iceland, on the other hand, are characterized by large lava flows and copious gas emissions but little ash. Volcanoes with both explosive and effusive characteristics are classified as "mixed."

Geologists now believe that there are at least 35 volcanoes with explosive potential located in the eastern United States and Alaska, the most dangerous being the Cascade Range and the Mono-Inya Craters of California (4). The Hawaiian volcanoes of Mauna Loa and Kilauea have major lava eruptions every few years; they have been intensively studied by geologists but pose little risk to human life compared with their explosive cousins on the U.S. mainland.

Temporal Patterns of Eruption

Volcanologists predict the general behavior of a volcano from available evidence on its geologic characteristics and past activity. For most volcanoes, statistical information on the timing of previous eruptive events for use in predicting future eruptions is sparse. For the few about which data exist, random eruption patterns (e.g., Mauna Loa, Hawaii), clustering (e.g., Kilauea, Hawaii), and the increasing probability of a violent eruption as time passes since the last eruption (e.g., Hekla, Iceland) have all been identified (1-3).

If the timing, size, and nature of a volcanic eruption could be accurately foretold, loss of life could be prevented by timely evacuation measures. In practice, this goal is remote. Warning of a reawakening of volcanic activity may be given by premonitory events such as small earthquakes or minor emissions of gas and ash over weeks or months. Specific monitoring techniques—including seismography and measurements of ground deformation, gaseous emissions, and thermal activity—may all be used. Cost consideration makes it impossible for all the world's active and dormant volcanoes to be regularly monitored by scientists. Alarmingly, severe eruptions have occurred at sites not even suspected of being volcanoes, e.g., Mount Lamington, Papua, where 2,942 people were killed in 1951 (3).

Ideally, hazard evaluation and emergency planning should be undertaken in all volcanic areas. A hazard evaluation made by U.S. Geological Survey scientists in 1978 (5) successfully predicted the impact of an eruption from Mount St. Helens, but the eruption that occurred on May 18, 1980, could be foretold only as a likely occurrence "possibly within

TABLE 1. Volcanic eruptions since A.D. 1600 that have caused over 8,000 deaths

Volcano	Date of eruption	Number killed	Lethal agent
Laki, Iceland	1783	9,350	Ashfalls destroyed crops and animals, causing starvation
Unzen, Japan	1792	14,300	70% killed by cone collapse; 30% by tsunami
Tambora, Indonesia	1815	92,000	Most deaths from starvation
Krakatoa, Indonesia	1883	36,417	90% killed by tsunami
Mt. Pelee, Martinique	1902	29,025	Pyroclastic flows
Ruiz, Colombia	1985	23,000	Mud flow

Source: Reference 3.

this century." For subsequent minor eruptions, however, geologists using monitoring techniques were able to improve their predictions to a day or less beforehand, providing important information for emergency-service personnel and for persons who continued to work in the volcano's vicinity.

Although advances in prediction and surveillance methods are likely, for the foreseeable future catastrophes such as that which occurred at Ruiz seem inevitable. Volcanic activity is unlikely to have lessened over the last thousands of years, whereas population expansion into volcanic areas has undoubtedly increased. History can still repeat itself in the form of events such as the destruction of Pompeii by Vesuvius in A.D. 79 when 16,000 people died. It is therefore increasingly important that effort in disaster mitigation is directed toward planning public health and other emergency measures while such predictive uncertainties remain.

Eruptive Hazards and Factors Influencing Morbidity and Mortality

The most common major lethal hazards in volcanic eruptions are pyroclastic flows and lahars, or mud flows (3). Unlike other hazards associated with eruptions, these provide little chance of survival to populations exposed, though the risk will depend on the size and nature of an eruption and local topographic factors, as well as on the proximity of populations. Asphyxiant gases will also be most dangerous near craters or fissures on or close to the volcano's flanks. Because gravity is crucial in determining the flows of solids and dense gases, low-lying areas and valleys near the volcano are generally at greatest risk. The public health consequences to be discussed first are those in the vicinity of the volcano.

Primary Factors

BLAST AND PROJECTILES

Blast is explosive force and is most destructive when accompanied by pyroclastic flow. Blast alone will shatter windows, leading to lacerations, and produce noise audible over long distances. Lethal rock fragments of varying sizes can be explosively ejected at any time. Large projectiles may damage houses and, if hot, start fires.

PYROCLASTIC FLOWS AND SURGES

These are mixtures of hot gases, ash, fine pumice, and rocks. Whether propelled by gravity or by explosive force over a crater rim, they travel at speeds of 50-150 km/hour, (31-91 mph) which—together with their content of solids—creates a powerful destructive momentum. Their temperatures are in the range of 600-900 C, though in some parts of the flow the temperature peak may be short-lived, so that survival under certain conditions is just barely feasible (6). The major lethal effects of pyroclastic flows lie in the density and temperature of the ash and rock fragments, which may cause asphyxia and inhalational injuries as well as body-surface burns. The thickness of deposited ash is less important, though persons closest to the volcano may be killed by blast or buried in ash and rock debris. The concentrations of toxic gases are probably relatively unimportant. In general, the chances of survival are small; for example, only two out of 22,000 people in the town of St. Pierre survived the pyroclastic flow at Mount Pelee in 1902 (Table 1).

The best opportunity to investigate the causes of death from pyroclastic flows was in the aftermath of the lateral-directed blast and pyroclastic flows from the May 18, 1980, eruption of Mount St. Helens. Despite official warnings and the establishment of restricted zones by local officials, over 100 people were within close range at the time of the eruption, including loggers going about their work. The lines of downed trees marked an abrupt cutoff of the maximal blast forces in an area of destruction extending as far as 27 km (17 miles) from the crater; 83% of people died in this zone (7). Examination of the bodies retrieved showed that 17 deaths had been caused by asphyxia and five by thermal injuries (two of the five decedents also had injuries to airways). Only three persons had been killed in trauma to the head from trees (two) or rocks (one). Three loggers located at a spot 19 km from the crater survived the direct blast with second- and third-degree burns affecting 33%-47% of body surface. Two died in the hospital from adult respiratory-distress syndrome induced by inhalation of hot, fine ash particles (8,9).

MUD FLOWS AND FLOODS

Mud flows (lahars) are frequent and deadly concomitants of eruptions. The heat from pyroclastic flows, lava, and steam blasts may melt glaciers and snow, whereas heavy rain may accompany ash eruptions. The water from these or other sources, e.g., crater lakes, will mix with ash and rock debris to form huge volumes of material with the consistency of concrete that is capable of engulfing everything in its path and destroying roads and bridges. After the May 18, 1980, eruption of Mount St. Helens, mud flows traveled down the Cowlitz and Toutle Rivers as far as 21 km (13 miles). The average speed of these flows along the valleys was only about 32 km/hour (20 mph), so that people living in their path downriver had time to be warned to escape, but those nearer the volcano were less fortunate. An estimated 85% died in the blast and mud-flow areas. No warning preceded the vast mud flow from the Ruiz, Colombia, volcano in 1985, which overwhelmed the town of Armero located 48 km (30 miles) away. The water in mud flows is sometimes scalding hot and can cause burns. Because river valleys are natural courses for mud flows, flooding may be a direct consequence of the filling of rivers and lakes. In addition, the deposited mud will alter the levels and courses of existing rivers, thereby posing a serious risk of future floods in the event of heavy rains. Floods may also be caused by avalanches into lakes or by melting ice and snow.

LAVA FLOWS

Lava flows from effusive volcanoes are very destructive of property, but their slow speed usually permits the inhabitants to escape in good time. Exceptionally, in 1977 a very fluid lake of lava at Nyiragongo, Zaire, drained suddenly, killing 300 people. In large effusive eruptions, diversion barriers and other methods of influencing the direction of a lava flow may be attempted.

EARTHQUAKES

Local earthquakes that frequently accompany eruptions may damage property and endanger life (see chapter on earthquakes). For example, the limited lava eruption of Mount Etna, Sicily, on December 25, 1985, was accompanied by several earth tremors, one of which destroyed a hotel in the vicinity and killed one person.

TSUNAMIS

These giant sea waves produced by subterranean shocks and explosions have caused heavy loss of life. Their occurrence is unpredictable, but they can devastate coastlines. The greatest loss of life from an eruption in recent times was caused by tsunamis after the eruption of Krakatoa in 1883 when over 36,000 people were drowned on the coasts of nearby Java and Sumatra.

GASES

Deaths caused by gases are uncommon compared with other volcano-associated fatalities, though it must be admitted that many such deaths may have gone unrecorded because accurate assessments of cause of death in volcanic eruptions are the exception rather than the rule. Tall volcanoes fortunately exert a "stack" effect, which—together with the heat and force of an eruption—usually results in dispersal of gases into the atmosphere. There are occasions, however, when gases may concentrate or be released at ground level.

The chief volatile emissions are water vapor, carbon dioxide, hydrogen sulfide, and sulfur dioxide, followed by hydrogen chloride, hydrogen fluoride, carbon monoxide, hydrogen, helium, and radon. Inorganic volatile material such as mercury may also be important in certain volcanoes, e.g., Kilauea. Organic volatile materials (polynuclear aromatic and halogenated hydrocarbons) may also be detected in eruptive plumes, particularly if the heat of the eruption has incinerated trees and other vegetation. Plumes from Mount St. Helens' eruptions were also found to contain appreciable quantities of carbonyl sulfide, carbon disulfide, and nitrogen dioxide (10).

Direct health effects. From the health viewpoint, volcanic gases can be classified as asphyxiants or respiratory irritants. Buildup of asphyxiant gases to lethal concentrations is likely only in the vicinity of a volcanic crater or fissure, whereas irritant gases may exert their effects in much lower concentrations for many kilometers downwind.

Animals grazing on many slopes of volcanoes in enclosed or low-lying regions have been asphyxiated, probably by carbon dioxide (CO_2) because it is denser than air. Hydrogen sulfide has also been reported to kill birds and cause sheep to be blinded. Children, geologists, and others roaming volcanic mountain areas are also at risk (11). On the Dieng Plateau, Java, in 1979, 142 people died while attempting to flee from a mild eruption, apparently overwhelmed by a powerful emission of CO_2, the source of which was less than 2 km away (11). In 1984 a cloud of CO_2 emitted from Lake Monoun situated in a volcanic field in Camaroon is believed to have killed 37 people (12). One hypothesis for this event is that it was caused by an overturning of the stratified waters of the lake in which CO_2 had slowly accumulated from a volcanic source (13). In 1986 a remarkable gas release occurred at Lake Nyos only 95 km (59 miles) to the northwest of Lake Monoun when about 1,700 people died. A huge amount of gas flowed at night into a mountainous, remote area north of the lake for as far as 20 km (12 miles) away; in a densely populated region the death toll would probably have been enormous. Hydro-geological tests of the water indicated that CO_2 was the sole—or at least major—gas released (14). Whether the gas was emitted by a volcanic eruption or by a mechanism hypothesized for Lake Monoun is unclear, though further investigations of the lake may provide an answer. The findings will have important implications for preventive measures at this as well as other lakes in volcanic areas.

widely studied in humans since it is a common air pollutant in industrialized countries for which recommended occupational and community exposure limits exist. Acute irritation to the lungs by sulfur dioxide may lead to effects ranging from subclinical constriction of the small airways in healthy adults (at concentrations in inspired air as low as 1 ppm for a few minutes) to frank asthma in susceptible persons. Exposure to low concentrations over long periods may result in an increase in respiratory illnesses in the general population. Regular exposure of the general population to volcanic plumes containing hazardous levels of sulfur dioxide and its aerosols has occurred at the Masaya Volcano, Nicaragua, during periods of very active degassing (13). With some volcanoes, e.g., San Cristobal in Nicaragua, the plumes of gas can travel down the slope to populated areas

on some days, depending on the weather conditions (13). During the eruptions of Kilauea, Hawaii, in 1983, gas sampling was undertaken to exclude a threat of sulfur dioxide to local populations (15). In the days after the eruption of La Soufriere in St. Vincent in 1979, Leus and his colleagues observed an increase in hospital admissions for children with a diagnosis of asthmatic bronchitis, but whether this was caused by inhalation of gases or ash (or both) is unclear (16).

Indirect health effects (e.g., acid rain). Rain falling through a volcanic plume will dissolve sulfur dioxide and other gases, becoming acidic in the process. At Masaya, Nicaragua, the rain water has been as acidic as pH 2.5-3.5, and people have complained that it has caused eye and skin irritation (13). In developing countries rain water is often collected from metal roofs and used for drinking. Health problems may arise if the acid rain dissolves metals or has a high fluoride content; the latter can occur if the plume contains a high concentration of hydrogen fluoride. Gases and acid rain will corrode machinery and cause damage to local crops.

ASHFALLS

The column of an explosive eruption will form a large plume that can travel in some instances for hundreds of kilometers downwind and cover a huge area with a layer of ash of various depths. An ashfall leaving a deposit 0.3 meters deep at a distance of 80 km (50 miles) from Mount St. Helens has occurred and could happen again. In such an event an unfavorable wind could cause ashfalls in some large cities west of the volcano. The eruptions in 1980 were on a much smaller scale: the maximum depth of ash was just over 4 cm in places (Figure 1) when the May 18 plume was blown eastward to the less populated areas.

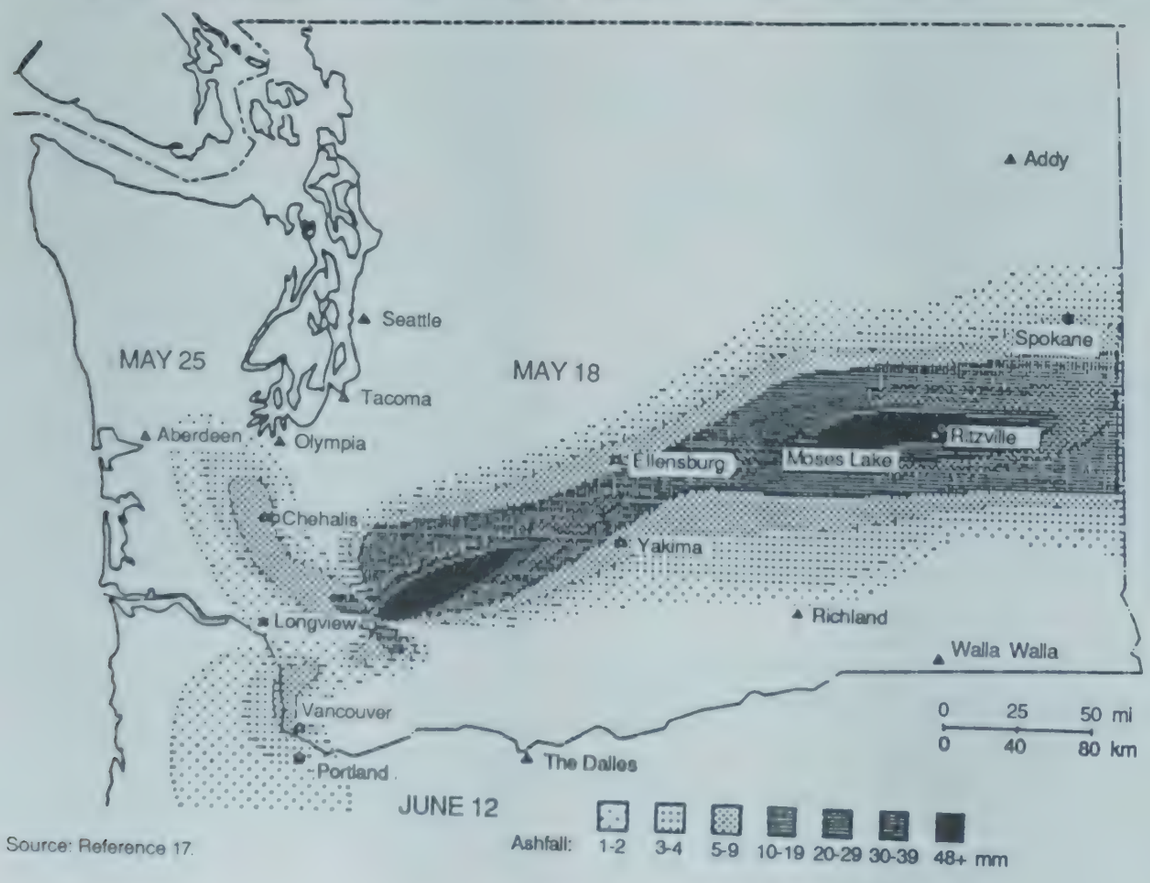
In the eruption plume, gases and other volatile material adheres to ash particles as they fall and, being readily soluble, will be washed off by rain into watercourses and onto crops. Icelandic eruptions, for example, are notorious for the high fluoride content of their ash. Freshly emitted ash may impart a sulfurous or pungent smell to the air, and the adherent volatile material probably adds to the irritant effects fine ash has on the lungs.

Volcanic ash can be produced from the explosion of old rock as well as from the release of pressure on the magma (fresh liquid rock) inside the volcano. The size of ash particles and their mineral composition vary among volcanoes and different eruptions (even of the same volcano). In general the finer particles in a volcanic plume fall farthest away. The particles may be small enough to be readily inhaled deep into the lungs; coarser particles will lodge in the nose or eyes and can also irritate the skin. In the ashfall from the Mount St. Helens' eruption on May 18, over 90% of the particles were within the respirable range in size, and ash continued to be resuspended in the air for a week afterwards until it rained. The elevated concentrations of fine ash in the air led to widespread anxiety about possible health effects (17).

DIRECT HEALTH CONSEQUENCES

Respiratory and ocular effects. Epidemiologic surveillance of trends in emergency room visits and hospital admissions

FIGURE 1. Ashfall after first three major eruptions of Mount St. Helens in 1980 (May 18, May 25, and June 12) and locations of Washington and Oregon hospitals in the Centers for Disease Control Epidemiological Surveillance System



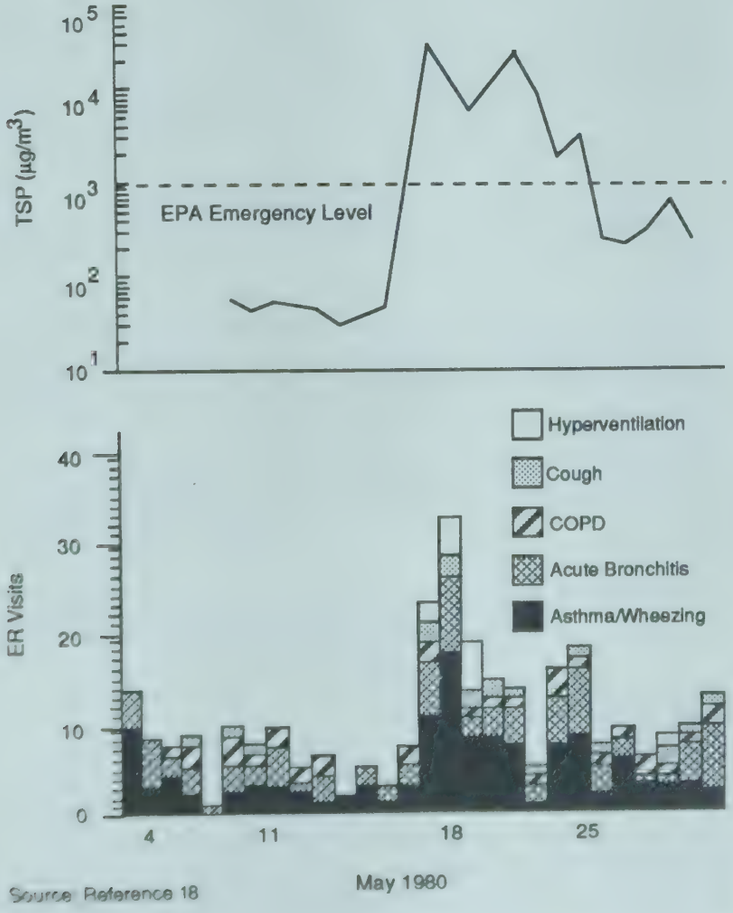
Source: Reference 17.

after each of the three main 1980 eruptions of Mount St. Helens revealed increases in the number of patients admitted for asthma and bronchitis (Figure 2) (17). In addition a household survey in Yakima showed that about a third of the patients with chronic lung disease who did not go to a hospital reported an exacerbation of their respiratory symptoms that was associated with the high levels of respirable ash in the ambient air (18). These patients would undoubtedly have been more severely affected if they had not heeded advice and their own common sense by staying indoors through the worst conditions. No deaths were attributed to the respiratory effects of the ash. Despite the absence of any good data, there is little doubt that the impact of similar eruptions in less-privileged countries would be greater.

Geologists do not routinely analyze ash for the presence of crystalline silica; therefore, the finding by health scientists that the Mount St. Helens' ash contained 3%-7% of this mineral by weight was first disputed and later confirmed (19). Outdoor occupational exposures for groups such as loggers and farm workers were potentially high enough to cause permanent lung damage (silicosis), but only if high exposure continued as a result of the volcano's erupting ash periodically over many years.

Eye irritation and minor corneal abrasions can result if ash particles enter the eye. These effects are not serious, but persons who wear contact lenses and persons heavily exposed to ash should wear protective eye gear.

FIGURE 2. Total suspended particulate (TSP) levels in ambient air before and after the May 18, 1980, eruption of Mount St. Helens and number of emergency room (ER) visits for asthma and bronchitis at the two major hospitals of Yakima, Washington, May 1980



Source: Reference 18

Toxic effects. The Mount St. Helens' ash was repeatedly tested for evidence of leachable elements, such as fluoride, but nothing abnormal was found. This result contrasts with findings in some Icelandic eruptions in which even a fine deposit of ash is sufficient to kill grazing animals. Elevated fluoride levels (as high as 9 ppm) were measured in streams after the Hekla eruptions of 1947 and 1970; water containing concentrations in excess of 1 ppm can cause bone disease (osteofluorosis) in humans if consumed over long periods (20). Water quality can also be adversely affected by acidic ash.

Mental health. The threat of an eruption or of having to cope with the aftermath of a major eruption may adversely affect mental health, according to investigators after the Mount St. Helens' eruptions (21).

INDIRECT HEALTH CONSEQUENCES

Since heavy ashfalls can cause widespread disruption, they can have serious public health implications.

Collapse of buildings. In North America, ordinary building codes and regulations do not usually take into account abnormally heavy roof loads. The flat roofs in modern public buildings and houses are particularly at risk of collapsing if the depth of accumulated ash exceeds a few inches, especially if rain falls on them (the rain greatly increases the weight). Old people and the infirm may be at special risk because they are advised to stay in their homes during an ashfall and therefore are unable to keep a susceptible roof clear of ash. Even the fittest individuals, however, are at risk of injury from falling off roofs. Flimsy dwellings in developing countries are especially prone to collapse under the weight of ash.

Lightning. Heavy lightning can frequently accompany an ash plume for kilometers from the volcano. Ground strikes can cause fatalities and fires.

Transportation and information dissemination. Virtually all transport can come to a halt in a heavy ashfall because of the impenetrable darkness and the damaging effect of ash on automobile, train, and plane engines. Cities could come to a virtual standstill for several days in the event of a massive ashfall, with implications for ambulance, fire, and police services. Automobile breakdowns and accidents due to slippery roads or poor visibility may pose serious problems. Telephone systems can become rapidly overloaded with anxious callers, and telephone switchgear may be damaged by the infiltrating ash. Radio and television transmission may suffer serious interference while the ash is falling.

Public utilities. Moist ash can be a good conductor of electricity and can cause short-circuiting of outdoor power equipment such as insulators, resulting in power outages. Engineers may be hampered in their repair tasks by transport problems. Many of the consequences of power outages are obvious; a less obvious consequence is that water supplies dependent on electrical pumping would be jeopardized.

Water supplies could also be restricted from fallen ash in reservoirs and rivers, which could clog filtration plants. Water quality could be impaired through turbidity and changes in pH. Sewage disposal machinery is rapidly overloaded and put out of action by abrasive ash.

Infectious hazards. The flooding and disruption of rivers and lakes could provide suitable conditions for the spread of leptospirosis and, in endemic areas, malaria.

General Risk Factors

General factors that contribute to the severity of the impact of the volcanic hazards listed include the following:

LACK OF PUBLIC AWARENESS

With population growth, human settlements have arisen in volcanic areas because of the economic benefits provided, e.g., from agricultural and logging activities and tourism. The obvious economic benefits have been perceived as outweighing the less tangible risks of volcanic activity, the latter tending to be ignored by local populations in developed and developing countries alike. Local officials responsible for community and industrial planning may also perceive risks associated with volcanic activity as insignificant until a major eruption occurs.

INADEQUATE MONITORING

Hazard evaluation by volcanologists has been undertaken for few of the world's volcanoes so far. Resources for the regular monitoring of volcanoes are limited, particularly in developing countries. Even when regular monitoring is being undertaken, dangerous eruptions can occur without adequate warning, e.g., Mount Mayon in 1984.

FALLIBILITY OF WARNINGS

Warnings given by volcanologists may not be acted on by government officials and local populations because of the fallibility inherent in predicting eruptions. Prolonged evacuation of populations can lead to severe socioeconomic disruption and much antagonism if an eruption fails to materialize.

VULNERABILITY OF POPULATIONS

The politicosocial development of a country may seriously limit the options available for adopting preventive measures. Residents of impoverished communities may feel that they have little choice but to remain where they are even when threatened by imminent volcanic activity. Disruption of communication links in such communities may also greatly add to loss of life after an eruption.

Preventive Measures

Some of the numerous public health consequences of major volcanic eruptions are common to other types of natural disasters, e.g., trauma from collapsing buildings, drownings, and deaths from exposure and starvation—still realities in developing countries despite modern international relief measures. Preventive measures outlined here are specific to volcanic eruptions (20,22). Guidance on general problems of the management of volcanic emergencies is also available (23).

Pre-Eruption Measures

As noted previously, the deadly eruptive phenomena responsible for the greatest loss of life are pyroclastic flows, pyroclastic surges, and lateral blasts (all health-related phenomena), mud flows, and tsunamis.

The only adequate preventive measures against these volcanic consequences are evacuation and the demarcation of restricted zones when an area is threatened by an eruption. In most instances the number of injured survivors in relation to the number of persons killed will be small; therefore, the role of medical treatment facilities in reducing the human scale of disaster must be limited, regardless of the influx of victims. Premonitory signs of renewed eruptive activity of an explosive volcano will therefore signal a public health emergency and the need for immediate planning measures involving public and occupational health officials at the earliest stage.

The measures in the vicinity of the volcano include:

- Evacuation and demarcation of at-risk areas, key preventive measures that essentially are decisions to be taken by government officials after consultation with volcanologists. Information from health officials also needs to be taken into account—e.g., on the feasibility of evacuating the sick, aged, and very young upon short notice. The health and welfare of persons evacuated temporarily or for long periods is also a public health matter. The safety of specific groups of workers who may be permitted into dangerous areas needs careful, unbiased assessment and dissemination of information on risks and benefits.
- Search and rescue plans for the dead and any marooned survivors after the eruption, including the sites for emergency field casualty stations and morgues and designation of their staff.
- Rehearsal of local hospital emergency plans for a sudden influx of victims with a) body-surface burns and lung damage from inhalation of hot ash and b) all kinds of trauma.
- Provision of emergency air-monitoring equipment for toxic gases.

In addition, planning for heavy ashfalls over a wide area, if applicable, should include arrangements for:

- Providing laboratory facilities for the collecting and analyzing of ash for leachable toxic elements and for monitoring drinking water quality. Specialist laboratories should be made available for measuring particle size and crystalline silica content of the ash, and for checking the bioavailability of toxic elements, e.g., in milk.
- Providing equipment for monitoring exposures to airborne ash in the community and in outdoor occupational groups.
- Stockpiling lightweight, disposable, high-efficiency masks, if indicated, for distribution to the public after an ashfall. Goggles and more robust respiratory protection may be needed for emergency workers and other outdoor workers.
- Preparing for possible temporary breakdowns of water supplies and sewage treatment plants, including ensuring adequate chlorination or the issuing of "boil water advisories."
- Maintaining emergency health services and hospitals.
- Providing emergency shelter and food relief.

Post-Eruption Measures

An emergency health team with medical, epidemiologic, and community health skills should be formed immediately after an eruption, and its membership should be planned before the emergency. One important function is collaborating with other agencies in an official disaster-coordinating

center and giving advice and information relating to all health aspects arising from the disaster. Field surveys may need to be conducted a) to collate epidemiologic information on the dead and survivors (e.g., cause of death and injury) and to collaborate with rescue teams and b) to make rapid assessments of health problems arising in the areas of heavy ashfall as soon as travel conditions permit.

Epidemiologic surveillance through a network incorporating hospitals and emergency rooms in affected areas will provide valuable information on the health impact of the disaster. Through such an information system the respiratory effects of the Mount St. Helens' ashfalls were first detected. The emergency and rescue services will perform many reactive tasks; further details can be found elsewhere (20,22). If a heavy ashfall has occurred, a key preventive measure is to maintain road blocks and adequate traffic controls in affected areas.

DISSEMINATION OF INFORMATION

At least 10-15 explosive eruptions occur throughout the world every year, but—with the exception of certain volcanoes, e.g., Merapi, Indonesia, which can erupt devastatingly every 15 years or so—they are uncommon or rare events in many parts of the world. Local communities may therefore be bewildered when told about a threatening eruption, and it will be essential to supply detailed information on what steps they should take before and after the eruption occurs.

Advice and equipment, including emergency warning systems, should be provided for people who are permitted to live or work in restricted or high-risk areas near a volcano. The mode of evacuation and the location of evacuation centers need to be specified. Similar provision should be made for those areas that might suffer heavy ashfall. Examples of brochures posted or given to households and workers for Mount St. Helens are given elsewhere (22). As in other types of disaster, radio and television will be essential for transmitting warnings and providing pre- and post-eruption advice.

Surveillance Measures

The shortcomings of facilities for monitoring volcanoes have already been noted. Accordingly, a good case can be made for an international team of experienced field volcanologists capable of traveling by invitation whenever an eruption threatens to rapidly set up monitoring systems and provide advice to local scientists and officials. Such collaboration will almost certainly be welcomed by scientists in developing countries and would go a long way toward mitigating volcanic disasters.

Critical Knowledge Gaps and Research Recommendations

There is hardly any aspect of volcanism in populated areas about which current knowledge cannot be increased (22). Unfortunately, international efforts to coordinate the management of volcanic disasters remain fragmentary, and there is little evidence that any international advances in preventive health and safety measures have been made since the Mount St. Helens' eruptions in 1980. A recently published monograph summarizing many of the health research activities in the wake of the Mount St. Helens' eruptions should serve as a valuable reference for future investigators dealing with other volcanoes (24). We especially need more information on health sequelae in developing countries in which the populations are most vulnerable and scientific studies are often difficult to perform. Considerable uncertainty is inherent in extrapolating from the experience of Mount St. Helens in the United States to countries in which the people may be underfed, may have inadequate housing and sanitation, and may be subject to a high incidence of fatal childhood respiratory infection.

An example is provided by the eruption of Ruiz, Colombia, in 1985. From the sparse reports in the literature, it would appear that few survive a massive mud flow, since virtually all people in the area are instantly engulfed. Nevertheless, numerous survivors were found for many days after the Ruiz eruption, as recorded by the world's media. We need to know more about how to reduce the vulnerability of populations to these predictable events, the best ways of rescuing survivors from mud flows, and the types of medical treatment that may be required before and after survivors arrive at the hospital. Such information has obvious implications for rescue services and saving lives and illustrates the importance of close collaboration between health and other emergency workers if advances in mitigating volcanic disasters are to be made.

Summary

Many major volcanic eruptions are preceded by premonitory events. Even so, geologists cannot usually predict with certainty the timing and size of an eruption. The first warning of reawakening of activity may therefore signal a public health emergency requiring from the outset the collaboration of health officials with other key groups in emergency planning.

The demarcation of restricted areas in the vicinity of the volcano is an essential first step in preventing loss of life from mud flows and pyroclastic flows; shorelines may also be at risk from tsunamis.

Pollution of air by respirable ash particles may exacerbate respiratory symptoms among patients with asthma and other chronic respiratory diseases. The greatest risk from asphyxiant gases is in topographic depressions lying within a few kilometers of a volcanic crater or fissure, but irritant gases such as sulfur dioxide may exacerbate respiratory disease and cause ecological effects from acid rain for kilometers downwind. Carbon dioxide is odorless, and the "rotten eggs" smell of hydrogen sulfide rapidly disappears if it is present in dangerous levels. Hydrogen sulfide also has irritant properties. Instruments for measuring concentrations of these gases should therefore be available in volcanic and geothermal areas where the potential for human exposure exists.

The best protection against the respiratory effects of fine ash is afforded by weather-proofed housing and lightweight, disposable, high-efficiency, industrial face masks to be worn when outdoors. Outdoor occupational groups may require heavy-duty industrial respirators and goggles. The public should avoid or evacuate areas at risk from gases. Appara-

tus for monitoring ambient and personal air levels for ash particles and gases may be essential for advising residents and workers.

The breakdown of public utilities, information systems, and transport; the needs of evacuees; and the damage caused by mud flows, floods, heavy ashfalls, earthquakes, and lightning can pose numerous direct and indirect health problems that remain poorly documented. An epidemiologic surveillance system linking emergency medical centers proved to be a valuable information resource after the Mount St. Helens' eruptions in 1980. Some of the health hazards are common to other natural disasters, but laboratory studies of ash to determine the presence of leachable toxic elements are essential so that advice can be given on contamination of water and food supplies for both humans and livestock. These studies should be performed immediately after an ashfall. Specialized laboratories should be available to measure particle characteristics, crystalline silica content, and other properties of the ash so that its short- and possibly long-term respiratory effects can be determined.

Advice on combating the numerous health consequences of eruptions should be encapsulated in handouts for the population and workers, and these handouts should be distributed before an eruption occurs. After an eruption, an emergency coordinating center is invaluable for the rapid and accurate dissemination of advice to officials and the public. Because the health and safety needs of populations in active volcanic areas are potentially so numerous, multidisciplinary approaches to planning and emergency response are essential.

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Hurricanes

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Background and Nature of Hurricanes

Factors That Contribute to Hurricanes

A hurricane is defined as a rotating wind system that whirls counterclockwise in the northern hemisphere, forms over tropical water, and has sustained wind speeds of at least 74 miles/hour (45.9 km/hour). This whirling mass of energy is formed when circumstances involving heat and pressure nourish and nudge the winds over a large area of ocean to wrap themselves around an atmospheric low. **Tropical cyclone** is the term for all wind circulations rotating around an atmospheric low over tropical waters. A **tropical storm** is defined as a cyclone with winds from 39 to 73 mph, and a **tropical depression** is a cyclone with winds less than 39 mph.

It is presently thought that many tropical cyclones originate over Africa in the region just south of the Sahara. They start as an instability in a narrow east-to-west jet stream that forms in that area between June and September as a result of the great temperature contrast between the hot desert and the cooler, more humid region to the south. Studies show that the disturbances generated over Africa have long lifetimes, and many of them cross the Atlantic. In the 20th century an average of 10 tropical cyclones each year whirl out across the Atlantic; six of these become hurricanes (1). The hurricane season is set as being June 1 through November 30. An "early" hurricane occurs in the 3 months before the season, and a "late" hurricane takes place in the 3 months after the season (2).

Hurricanes are well-organized. The 10-mile-thick inner spinning ring of towering clouds and rapid upper motion is defined as the hurricane's eyewall; it is here that condensation and rainfall are intense and winds are most violent. Harbored within the eyewall is the calm eye of the hurricane—usually 10-20 miles across—protected from the inflowing winds and often free of clouds. Here, surface pressure drops to a minimum, and winds subside to less than 15 mph. Out beyond the eyewall, the hurricane forms into characteristic spiral rain bands, which are alternate bands of rain-filled clouds. In the typical hurricane, the entire spiral storm system is at least 1,000 miles across, with hurricane-force winds of 100 miles in diameter and gale-force winds of 400 miles in diameter. A typical hurricane liberates about 100 billion kilowatts of heat from the condensation of moisture, but only about 3% of the thermal energy is transferred into mechanical energy in the form of wind. Sustained wind speeds up to 200 mph have been measured, but winds of about 130 mph are more typical. It is estimated that an average hurricane produces 200 billion tons of water a day as rain (1).

Most of the flooding associated with hurricanes does not come from the intense rain but from what is known as the "storm surge." The winds and low pressure around the hurricane eye tend to raise the level of the ocean 1-2 feet. When this dome of water, perhaps 50 miles across, moves into shallow coastal water, the decreasing water depth transforms it into a storm surge that can rise high above normal sea level, bringing the seas well inland. Depending on the strength of the storm and local bottom conditions where the storm comes ashore, the storm surge may cause the sea to rise as much as 20 feet higher than normal. The storm surge is superimposed on normal, astronomically based tides. In turn, wind waves are superimposed on the storm surge. The worst circumstance is to have the storm surge strike the coast at the time of high tide. During hurricane Camille in August 1969, one of the strongest U.S. hurricanes of this century, the storm surge was estimated to be 30 feet high (1).

The destructive power of a hurricane is determined by the way storm surge, wind, and other factors are combined. Hurricane forecasters have developed a five-category disaster potential scale to make comparisons easier and to make the predicted hazards of approaching hurricanes clearer. Category 1 is a minimum hurricane, category 5 a maximum hurricane (Table 1)(2).

Hurricanes generally move along their path at speeds of less than 20 mph, especially in the formative stages, but on rare occasions one will race along up to 60 mph. Hurricanes have an initial westward-moving flow. If they drift far enough northward, they can be caught by the prevailing westerly wind of the mid-latitudes and redirected to the east or northeast. Their direction is also influenced by other large-scale weather conditions. A southeastward-advancing cold front, for example, can stall a northwestward-moving hurricane off the U.S. coast and perhaps nudge it back out to

TABLE 1. Saffir/Simpson hurricane scale ranges

Scale number (category)	Central pressure millibars	Central pressure inches	Winds (mph)	Surge (in feet)	Damage
1	≥980	≥28.94	74-95	4-5	Minimal
2	965-979	28.50-28.91	96-110	6-8	Moderate
3	945-964	27.91-28.47	111-130	9-12	Extensive
4	920-944	27.17-27.88	131-155	13-18	Extreme
5	<920	<27.17	>155	>18	Catastrophic

Source: Reference 2.

TABLE 2. Number of hurricanes (direct hits) affecting the United States, by individual states, 1900-1982, according to Saffir/Simpson hurricane scale

Area	Catagory number 1	2	3	4	5	All	Major hurricanes (>category 3)
U.S. (Texas to Maine)	48	33	40	13	2	136	55
Texas	9	9	8	6	0	32	14
(North)	4	3	2	4	0	1	6
(Central)	2	2	1	1	0	6	2
(South)	3	4	5	1	0	13	6
Louisiana	5	5	7	3	1	21	11
Mississippi	1	1	4	0	1	7	5
Alabama	4	1	4	0	0	9	4
Florida	16	14	15	5	1	51	21
(Northwest)	9	6	5	0	0	20	5
(Northeast)	1	7	0	0	0	8	0
(Southwest)	5	3	5	2	1	16	8
(Southeast)	4	10	7	3	0	24	10
Georgia	1	4	0	0	0	5	0
South Carolina	5	4	2	1*	0	12	3
North Carolina	9	3	6	1*	0	19	7
Virginia	1	1	1*	0	0	3	1*
Maryland	0	1*	0	0	0	1*	0
Delaware	0	0	0	0	0	0	0
New Jersey	1*	0	0	0	0	1*	0
New York	3	0	4*	0	0	7	4*
Connecticut	2	1*	3*	0	0	6	3*
Rhode Island	0	1*	3*	0	0	4*	3*
Massachusetts	2	1*	2*	0	0	5	2*
New Hampshire	1*	0	0	0	0	1*	0
Maine	4	0	0	0	0	4	0

*Indicates all hurricanes in this category were moving at >30 mph.

Note: State totals do not equal U.S. totals, and Texas and Florida sectional totals do not equal state totals.

Source: Reference 3.

sea. However, hurricanes may follow very erratic paths, and it is unsound to generalize about what direction they will take.

Once a hurricane moves over the mainland, its supply of evaporated water from the ocean surface is cut off and its power eventually diminishes, but not before it dumps enormous quantities of rain on the land below (1).

Trends over Time

In the period 1900-1982, 136 hurricanes struck the United States directly; 55 of these were of at least category-3 intensity (Table 2). Florida felt the effects of both the highest number (51) and the most intense of these storms, with Texas, Louisiana, and North Carolina following in descending

TABLE 3. Number of hurricanes of various categories to strike the United States each decade of the 20th century

Decade	Category					All	Major
	1	2	3	4	5	(Categories 1-5)	(Categories 3-5)
1900-1909	4	4	4	2		14	6
1910-1919	8	3	5	3		19	8
1920-1929	7	2	3	2		14	5
1930-1939	4	5	6	1	1	17	8
1940-1949	7	8	7	1		23	8
1950-1959	8	1	7	2		18	9
1960-1969	4	5	3	2	1	15	6
1970-1978	5	1	3			9	3
TOTAL	**47**	**29**	**38**	**13**	**2**	**129**	**53**

Note: Only the highest category to affect the United States has been used.

Source: Updated from Hebert and Taylor 1975.

order (3). The number of hurricanes of various categories to strike the United States in each decade from 1900 through 1978 is shown in Table 3. Based on 100 years of record keeping on hurricanes, the National Weather Service has observed that on the average, a category 4 or higher hurricane strikes the United States once every 5 years (2). However, in the period 1950-1978, both the number and intensity of land-falling hurricanes decreased sharply. The expected number of hurricanes in the period 1958-1977 was 34, but only 14 were observed. A study by Hebert and Taylor in 1975 showed that as of 1970 almost 80% of coastal residents from Texas to Maine had never experienced a direct hit by a major hurricane. Many of these 28 million residents had moved to coastal sections during the preceding 20 years. This below-average trend continued through 1984 (4). During that time, rapid growth of the coastal population continued.

In 1985 the number of hurricanes striking the U.S. mainland was more in keeping with the average pattern. In that year six hurricanes struck the U.S. mainland; two were of category 3 or higher. Hurricane Gloria was measured as a category 5 over the Atlantic Ocean; it struck Cape Hatteras, North Carolina, as a category 4, and went on to strike Long Island as a category 3. Hurricane Elena struck Florida, Alabama, and Mississippi as a category 3. Many people considered 1985 an abnormally devastating year for hurricanes because they had been lulled into thinking that major hurricanes seldom struck the U.S. mainland because of their experience during a below-average period. However, a specialist at the National Weather Service is quick to point out that 1985 more closely approximated the average (H. Gerrish, personal communication).

Factors Influencing Morbidity and Mortality

Although hurricane winds do much damage, the wind is not the biggest killer in a hurricane. Most victims die by drowning. The flooding that accompanies a hurricane, for the most part, does not come from the intense rain but from the storm surges. The National Weather Service estimates that storm surges cause nine of every 10 hurricane-associated fatalities (1).

Table 4 lists the deadliest hurricanes to strike the United States in this century (1900-1982). The 12 deadliest hurri-

TABLE 4. The deadliest (25 or more deaths) U.S. hurricanes, 1900-1982, in descending order of severity

	Hurricane	Year	Category	Deaths
1.	Texas (Galveston)	1900	4	6,000
2.	Florida (Lake Okeechobee)	1928	4	1,836
3.	Florida (Keys/S. Texas)	1919	4	600-900†
4.	New England	1938	3*	600
5.	Florida (Keys)	1935	5	408
6.	AUDREY (Louisiana/Texas)	1957	4	390
7.	Northeast U.S.	1944	3*	390§
8.	Louisiana (Grand Isle)	1909	4	350
9.	Louisiana (New Orleans)	1915	4	275
10.	Texas (Galveston)	1915	4	275
11.	CAMILLE (Miss./La.)	1969	5	256
12.	Florida (Miami)	1926	4	243
13.	DIANE (Northeast U.S.)	1955	1	184
14.	Southeast Florida	1906	2	164
15.	Mississippi/Alabama/Pensacola	1906	3	134
16.	AGNES (Northeast U.S.)	1972	1	122
17.	HAZEL (S.C./N.C.)	1954	4*	95
18.	BETSY (Fla/La.)	1965	3	75
19.	CAROL (Northeast U.S.)	1954	3*	60
20.	Southeast Florida/La.-Miss.	1947	4	51
21.	DONNA (Fla./Eastern U.S.)	1960	4	50
22.	Georgia/Carolinas	1940	2	50
23.	CARLA (Texas)	1961	4	46
24.	Texas (Velasco)	1909	3	41
25.	Texas (Freeport)	1932	4	40
26.	South Texas	1933	3	40
27.	HILDA (Louisiana)	1964	3	38
28.	Southwest Louisiana	1918	3	34
29.	Southwest Florida	1910	3	30
30.	CONNIE (North Carolina)	1955	3	25
31.	Central Louisiana	1926	3	25

* Moving >30 mph.
† Over 500 of these lost on ships at sea.
§ 344 of these lost on ships at sea.

Note: Information for earlier years:

Year	Location	Deaths
1881	Georgia/South Carolina	700
1893	Louisiana	2,000
1893	South Carolina	1,000-2,000

Source: Reference 5.

canes were all classified as the equivalent of category 4 or higher. All but two of the 31 deadliest hurricanes were major hurricanes. The exceptions were the inland flood-producing hurricanes Agnes and Diane. In association with the other 29 hurricanes, the death tolls are primarily a result of the 15- to 20-foot rise of the ocean (storm surge) associated with these hurricanes (5). During hurricane Camille, the storm surge was estimated to be 30 feet high; 256 persons died in that hurricane. Among them were 23 of 24 persons at Pass Christian, Mississippi, who refused to evacuate and held a "hurricane party" instead. The three-story apartment they were in was obliterated by the storm surge, leaving nothing but the foundation. The one survivor was buffeted about in the debris-strewn waters before eventually being swept into a tree top, where she was found and rescued the next morning. A storm surge in the Louisiana bayou country in October 1893 killed 2,000 people. A storm surge on Lake Okeechobee, Florida, in September 1928 broke the dikes and claimed 1,836 victims. A storm surge struck the Florida Keys in 1935, leading to a loss of 408 lives; one that struck Louisiana in 1935 left a death toll of 390 (1).

The decreased death tolls in the past 20 years may reflect the below-average number of major hurricanes striking the most vulnerable areas. However, as the incidence and intensity of land-falling hurricanes reverts to the average pattern, the death tolls may again rise because of increased coastal growth. If warnings are heeded and preparedness plans developed, the death toll can be reduced, but large property losses are inevitable.

The six hurricanes that struck the U.S. mainland in 1985 are shown in Table 5 by category and number of associated deaths (5). It is interesting to note that Juan, only a category 2 hurricane, had the highest death toll. Most of these were drowning deaths associated with the flash flooding that resulted from the heavy rainfall that accompanied the hurricane.

More detailed information on risk factors contributing to morbidity and mortality from coastal and flash flooding is included in the chapter on floods.

TABLE 5. Hurricanes striking U.S. mainland in 1985, by category and number of associated deaths

1985 Hurricanes (Areas affected)	Category	Number of deaths
Gloria (North Carolina to Maine)	4 - Cape Hatteras 3 - Long Island	8
Elena (Florida, Louisiana, Alabama, Mississippi)	3	4
Juan (Louisiana, Mississippi, Florida Panhandle)	2	12
Kate (Florida Panhandle, Georgia)	2	5
Danny (Louisiana)	1	1
Bob (Alabama)	1	0

Source: Reference 5.

Public Health Implications

Prevention and Control Measures

The principal steps to follow for preventing death and injury associated with hurricanes are:

1. To identify meteorologic precursors of hurricanes and track their course and potential development into hurricanes.

   The National Hurricane Center of the National Oceanic and Atmospheric Administration in Coral Gables, Florida, has developed a highly sophisticated system for identifying and tracking tropical cyclones. A tropical cyclone may first be identified by satellite and then tracked by radar. In addition, computer portrayals of the behavior of hurricanes are valuable to atmospheric scientists in studying the inner workings of these tropical storms. Information gained during flights by highly instrumented aircraft into hurricanes helps to form a more complete picture of storm conditions.

2. To issue early hurricane warnings to provide for timely evacuation when indicated.

   The highly sophisticated tracking system of the National Hurricane Center probably provides a better early-warning system for hurricanes than is available for any other type of weather-related disaster. Although hurricanes sometimes follow an erratic course, the predictions of when and where they may strike are certainly more reliable than those for flash floods and tornadoes. The hurricane warnings are communicated through the National Weather Service Communication Network, and the information is readily available to the media. Radio and television usually give high visibility to such events and can be very effective in alerting the public to the potential danger of a hurricane.

3. To enforce stringent land-use management practices and building codes in high-risk areas.

   Unfortunately, indiscriminate development permitted along the Atlantic and Gulf coasts in the past 20 years has lead to densely populated high-risk areas. However, more stringent building codes have been introduced in many areas, and most building codes require a building to sustain winds of 130 mph. Florida recently changed its building code to require structures to withstand winds of 140 mph rather than 130 mph.

4. To develop emergency contingency plans in high hurricane-risk areas to provide for an orderly evacuation and adequate shelter capacity for evacuees.

   Evacuation from coastal areas is a critical preventive measure for hurricanes because of the great danger from storm surges. Most coastal communities have contingency plans, and Texas and Florida have very well-developed plans. There has been some concern that the people may not respond to evacuation recommendations because they have been lulled into a false

sense of security because of the below-average number of dangerous hurricanes that have struck the United States in the past 20 years. However, most people chose to evacuate during the 1985 hurricanes, and some people had to evacuate twice during hurricane Gloria because of the erratic path of that hurricane.

Because of heavy development and increased population density on some of the offshore islands along the Atlantic and Gulf Coast, special attention must be given to the time necessary to provide an orderly and successful evacuation across the causeways connecting the islands to the mainland.

For other preventive and control measures relating to flooding and high winds, the reader is directed to the chapters on floodings and tornadoes.

Surveillance

The National Hurricane Center not only has a very effective tracking system for hurricanes but also collects and analyzes data on hurricanes from all over the world. It routinely prepares and disseminates written reports based on these data.

For surveillance activities related to morbidity and mortality from hurricane associated floods, the reader is referred to the chapter on floods.

Research Recommendations

1. Because the meteorologic factors contributing to hurricanes have been studied in some detail, a good deal of information is available on this phenomenon. However, more information is needed on factors influencing the variable pattern of incidence and intensity of hurricanes striking the United States over time.

2. Emergency contingency plans for various coastal communities are tested and should be evaluated when hurricanes strike a particular community. The plans should be revised on the basis of these findings.

3. Does protection from wind speed also protect buildings from storm surges? Can those buildings designed to withstand winds of 130 mph also withstand sea surges of 25-30 feet? Research is needed to address these questions.

Other research recommendations related to hurricane associated flooding are addressed in the chapter on floods.

Summary

In the period 1900-1982, 136 hurricanes directly hit the U.S. mainland. Fifty-five of these storms were category 3 or higher. Florida experienced both the highest number of hurricanes and those of greatest intensity. The hurricane season is defined as June 1 through November 30. In the period 1950-1978, both the number and intensity of land-falling hurricanes in the United States decreased sharply. These circumstances created a false sense of security with regard to dangers involved in living near coastal areas and led to massive development and increased population density in potentially high-risk areas. In 1985 six hurricanes struck the United States; two of these were category 3 or higher. The National Weather Service considers the 1985 incidence more in keeping with the average pattern than that seen in the previous several years.

Although hurricane winds do much damage, the wind is not the biggest killer in a hurricane. Most victims die by drowning. The flooding that accompanies a hurricane, for the most part, comes not from the intense rain but from storm surges. The National Weather Service estimates that storm surges cause nine of every 10 hurricane-associated fatalities.

Critical prevention measures are in place for limiting morbidity and mortality associated with storm surges from hurricanes if people will follow warnings and orders to evacuate. These preventive measures involve early detection and tracking of hurricanes so that timely warnings can be issued and orderly evacuation from threatened coastal areas carried out. The National Hurricane Center in Coral Gables, Florida, has developed a very sophisticated system for identifying and tracking hurricanes, and the warnings issued by the Center are timely and have a high success rate for accurately predicting the time and place a hurricane may strike the United States. Most coastal communities have developed emergency contingency plans that provide for evacuating and sheltering people. The mortality associated with the flash flooding that often accompanies a hurricane is more difficult to control because technology for accurately predicting the exact time and place a flash flood might occur is still limited.

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Tornadoes

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Background and Nature of Tornado-Related Disasters

Meteorologic Description of the Generation and Characteristics of Tornadoes

Circular motions of air within the earth's atmosphere are called vortices; they vary in size from dust devils, through tornadoes and waterspouts, to hurricanes and cyclones (1). In the northern hemisphere, the earth's rotation causes the vortex wind to be deflected in a counterclockwise spiral around a low-pressure center. This phenomenon is reversed in the southern hemisphere, where vortices move in a clockwise fashion.

Waterspouts are similar to tornadoes except that they occur only over oceans and lakes. Because of their less-vigorous rotation and reduced human exposure, they are usually less destructive than tornadoes. One reason tornadoes can be so damaging is their tremendous pressure. Within the "eye" of a tornado the pressure may be $>$ 100 millibars below that of the surrounding air. In comparison, hurricanes can be characterized by the same differential except that it may be spread across hundreds of miles.

For a tornado to form, layers of air of different temperatures, moisture, density, and wind flow are required. Tornadoes usually develop within intense thunderstorm clouds (cumulonimbus) when certain weather conditions exist. These conditions include unseasonably warm, humid air at the surface, with cold air at the middle atmospheric levels, and a strong upper-level jet stream (2). The energy transformation necessary to generate a tornado has been hypothesized to result from the effect of thermally induced rotary circulations, or from the effect of converging rotary wind. The most likely explanation entails a combination of both thermal and mechanical forces (3).

Once a tornado is formed, it is maintained and even strengthened by the flow of warm air up the center of the vortex (4). As a tornado moves along the ground, destruction within its path is virtually absolute. Only reinforced concrete or steel can withstand the wind speeds and pressure differentials characteristic of tornadoes. Tornado outbreaks from a weather system are classified as follows: small, six to nine tornadoes; medium, 10-19 tornadoes; and large, $\geq$ 20 tornadoes (5).

Patterns for Occurrence of Tornadoes

Reports of incidence of tornadoes come frequently from the United States but also from countries all around the world (6,7). The U.S. tornado frequency appears to be no larger than that of other industrial counties in the middle latitudes (8). The biggest difference may be the occurrence of extremely large and intense tornadoes in the United States (8). Analysis of tornadoes in the United States in the period 1950-1976 showed an average annual incidence of 654 (9). In 1982, 1,027 tornadoes struck in the United States; this was only the second time in recorded history that the U.S. annual tornado count exceeded 1,000 (10).

The forward speed of tornadoes has been observed to range from almost no motion to 70 miles/hour (mph) (112 km/hour). The average forward speed for tornadoes is 40 mph, a relatively slow rate compared with rotary speeds (3,11). Most (63%) tornadoes in the United States have a rotary speed of $\leq$ 112 mph, while only 2% have speeds $>$ 206 mph (2).

The three influential topographic effects on tornado incidence and speed are elevation, slope, and roughness. Tornadoes tend to decrease in number and intensity as elevation increases (12). Tornadoes should not occur in mountainous areas as frequently as they do in flatter, smoother locales (13). However, examples can be cited in which tornado outbreaks have little regard for terrain (14).

With reference to direction of travel, statistics for 3,090 tornadoes in the United States from 1930-1950 showed that 61% moved from the southwest, 16% from the west, and 11% from the northwest (15). Often, several tornadoes are generated from the same front and move in parallel directions.

An important characteristic of a tornado is the length of its path. An early analysis of nonskipping tornadoes showed a mean path length of 2 miles; 95% of all tornadoes have a path length of less than 8 miles (16). An analysis of all tornadoes (skipping and nonskipping) in the United States in the period 1950-1975 showed a mean path length of 3.3 miles

(17). Several tornadoes with continuous paths of ≥ 200 miles have been documented (15). On May 26, 1917, a tornado traveled 293 miles across Illinois and Indiana and lasted 7 hours and 20 minutes (3).

The average path width of tornadoes in the United States is about 1,200 feet (366 meters), with a documented range of 9 feet to 8 miles (15). In 1982 the average tornado had a track approximately 2 miles long and 50 yards wide, affecting about 0.06 square miles (10).

The Fujita and Pearson Tornado Scale ranks tornadoes according to their ferocity, as measured by their speed, path length, and path width (Table 1). Of the 17,659 tornadoes in the United States in the years 1950-1976, 14,409 had information recorded about both speed and path characteristics. Of these tornadoes 62% were weak (F0 or F1), 36% were strong (F2 or F3), and only 2% were violent (F4 or F5) (9).

With respect to seasonality, tornadoes have occurred during all months, although they are most frequent in spring (18). With reference to geographic distribution, tornadoes have been reported in all 50 states (17). From 1916 to 1950, 5,204 tornadoes occurred in the United States. The states having the largest numbers of documented tornadoes during this period were Kansas (618), Iowa (498), Texas (481), Oklahoma (399), and Arkansas (324). Nevada and Rhode Island each reported only one tornado (15).

TABLE 1. Fujita and Pearson Tornado Scale

Scale	Speed	Path length	Path width
	(mph)	(miles)	
0	≤72	<1.0	≤17 yards
1	73-112	1.0-3.1	18-55 yards
2	113-157	3.2-9.9	56-175 yards
3	158-206	10.0-31.0	176-556 yards
4	207-260	32.0-99.0	0.34-0.9 miles
5	261-318	100-315	1.0-3.1 miles
6	319-380	316-999	3.2-9.9 miles

Source: Reference 9.

Public Health Impacts: Historic Perspective

Despite their relatively narrow paths, tornadoes are the most lethal and most violent of all natural atmospheric phenomena. This may be largely because of their wind velocity, sudden generation, and erratic movement. The destruction caused by tornadoes results from the combined action of their strong rotary winds and the partial vacuum in the center of the vortex (3). For example, while passing over a building, the winds twist and rip at the outside at the same time the abrupt pressure reduction in the tornado's eye causes explosive pressures inside the building. Walls collapse or topple outward, windows explode, and the debris of this destruction is driven as missiles through the air. The amount of mechanical energy available is perhaps best illustrated by example. In 1931 a tornado in Minnesota carried an 83-ton railroad coach and its 117 passengers 80 feet through the air and dropped them in a ditch (3).

In the last 50 years, tornadoes have been responsible for more than 9,000 deaths (19). From 1916 to 1950, tornadoes resulted in 7,931 deaths in the United States, for a yearly average of 227 deaths (15). In the United States tornadoes killed 530 persons in the period 1962-1966 (20). For the 10-year period 1965-1974, more than 1,400 tornado-associated deaths were reported in the United States. During this same period there were 8,000 tornadoes (21). In the United States in 1984, tornadoes resulted in 122 deaths and $1 billion in property damage (22). Overall, tornado fatalities have decreased in recent years, while numbers of tornadoes reported have increased (17).

In terms of public health impact, mortality figures represent only the tip of the iceberg. For example, on the evening of March 28, 1984, a series of tornadoes in North Carolina and South Carolina resulted in over 1,000 casualties (people killed and injured) and $100 million in property damage. Medical records for 955 injured persons showed that 59 (6%) were killed, 256 (27%) hospitalized, and 640 (67%) treated and released (23). Data pertaining to tornado-associated mortality and morbidity occurring in the United States in 1970-1980 are shown in Table 2. In the 1970s tornadoes resulted in death to 856 persons, injury to 22,012 persons, and need for emergency care (shelter, clothing, food, or medical supplies) for 909,605 persons. Approximately 4% of all injuries sustained were fatal. For every person injured or killed, approximately 44 others required some type of emergency care.

TABLE 2. Public health impact of tornadoes, United States, 1970-1980

	Number of persons		
Year	Killed	Injured	Received emergency care
1970-1971	145	1,823	34,451
1971-1972	22	653	12,833
1972-1973	31	993	18,822
1973-1974	412	10,574	189,817
1974-1975	48	688	38,488
1975-1976	40	1,213	24,315
1976-1977	11	369	17,960
1977-1978	21	448	23,165
1978-1979	100	4,209	490,316
1979-1980	26	1,042	59,438
TOTAL	**856**	**22,012**	**909,605**
MEAN	86	2,201	90,961

Source: American Red Cross Data, as reported in: Gordon PD. Special statistical summary - deaths, injuries, and property loss by type of disaster. 1970-1980. Washington, D.C.: Federal Emergency Management Agency, 1982.

When dealing with statistics, it is important to realize the proportion represented by disasters associated with single severe events. Analysis of 14,600 tornadoes between 1952 and 1973 showed that 497 tornadoes caused 2,575 fatalities. Of these 497, 235 each caused only a single fatality, while 26 tornadoes caused 1,180 deaths (24). In decreasing order of importance, the largest tornado disasters in the United States follow: March 18, 1925 (606 killed in Illinois); April 2-7, 1936 (402 killed in Mississippi, Alabama, and Georgia); April 3, 1974 (307 killed in southern and midwestern states); April 11, 1965 (272 killed in Indiana, Ohio, Michigan, Illinois, and Wisconsin); and March 21-22, 1952 (229 killed in Arkansas, Tennessee, Missouri, Mississippi, and Alabama)(25).

The tri-state tornado of March 18, 1925, was the largest and strongest tornado in the recorded history of the United States. The storm traveled 219 miles at 60 mph, and 35% of all injuries reported were fatal (26). In terms of number of tornadoes, length of tracks, total area affected, and amount of damage, the April 3-4, 1974, tornado outbreak was the most devastating (5). For all the tornadoes in this outbreak, the mean path length was 18.7 miles, whereas the mean path length for all tornadoes in 1973 was 4.7 miles. The tornadoes in the 1974 outbreak had total accumulated paths of 2,014 miles and resulted in 315 deaths. The Palm Sunday outbreak on April 11, 1965, involved 31 tornadoes whose paths totaled 853 miles; 256 persons were killed. This tornado disaster caused 1,183 persons to be hospitalized; 6,142 were injured. There were 7,512 permanent homes and 2,901 mobile homes destroyed (27).

The probability that a tornado will strike any specific location is extremely small. For areas most frequently subjected to tornadoes, the probability is 0.0363, or about once every 250 years (3). However, the literature does contain reports of exceptions. Oklahoma City has been struck by tornadoes 26 times since 1892. Codell, Kansas, was struck in 1916, 1917, and 1918, all on May 20. On March 21, 1932, a tornado struck Sylacauga, Alabama, and killed the second wife of Luther Kelley. Mr. Kelley had married this woman after his first wife was killed when a tornado struck Sylacauga on May 28, 1917 (5).

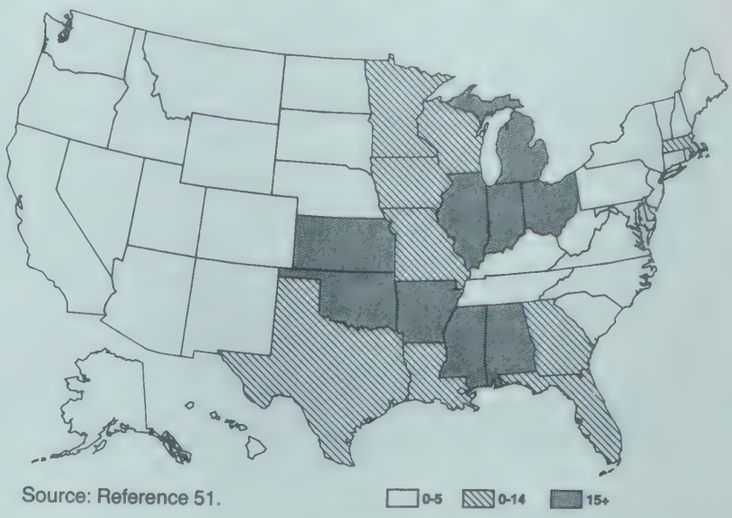
Factors Influencing Morbidity and Mortality

Environmental

An important risk factor appears to be geographic location within the United States (18). Both the occurrence of tornadoes and the incidence of deaths vary by state. Researchers from the National Weather Service and academia have published numerous maps that describe these distributions. Two such maps included here as examples are based on 1953-1969 data from the National Atmospheric and Oceanic Administration's (NOAA) Environmental Data Service. (Figures 1 and 2). Mean annual numbers of tornadoes/10,000 square miles, by state, are shown in Figure 1. In this period, 18 states averaged ≤ 1.5 tornadoes/10,000 square miles. The states experiencing the fewest tornadoes during this 17-year-period included Rhode Island (zero), Alaska (one), Hawaii (four), and Nevada (seven). Fifteen states averaged more than three tornadoes/10,000 square miles, with the highest rates occurring in Oklahoma (8.8), Kansas (6.4), and Indiana (6.1). The states with the largest numbers of tornadoes were Texas (1,578), Oklahoma (1,042), and Kansas (876).

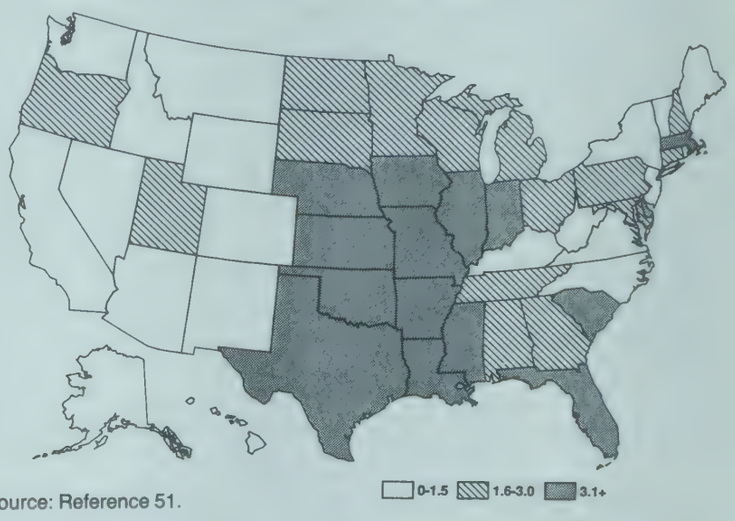
Total deaths/10,000 square miles, by state, are shown in Figure 2. In the 17-year period, 14 states had no fatalities. Nine states sustained 15 or more fatalities/10,000 square miles, with the largest rates occurring in Indiana (40), Michigan (37), and Mississippi (35). The states with the highest numbers of deaths were Texas (234), Michigan (218), and Mississippi (169). Overall, the greatest risk of fatalities may not be where the largest number of tornadoes occur but where a large population at risk is combined with a high incidence of tornadoes (28).

FIGURE 1. Mean annual numbers of tornadoes per 10,000 square miles, by state, United States, 1953-1969



Source: Reference 51.

FIGURE 2. Total mortality from tornadoes per 10,000 square miles, by state, United States, 1952-1969



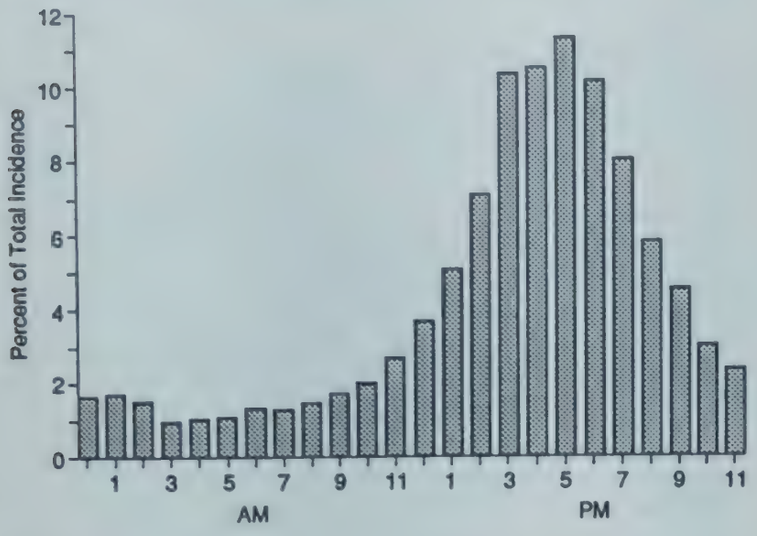
Source: Reference 51.

Numerous other maps are based on such indices as tornado ground-contact path areas, mean number of tornadoes, total path lengths/one degree square, and average fractional square miles ravaged by tornadoes. Not surprisingly, these maps are meteorologically rather than epidemiologically based and usually focus on large areas—either states or regions. It appears that more detailed assessments specific to public health issues are needed.

Other pertinent factors for assessing risk are the apparent, nonrandom diurnal and monthly distributions of tornado incidence. Information provided in Figure 3 depicts the diurnal distribution of tornadoes (16,860) that occurred in the United States from 1950 to 1970. Approximately 42% of all tornadoes occurred from 3:00 p.m. to 6:00 p.m. During maximum sleeping hours (12:00 midnight to 6:00 a.m.), just under 10% of the tornadoes occurred. This trend in the incidence of tornadoes with respect to time of day may be directly associated with adverse weather. This late afternoon maximum incidence may be associated with the time of maximum solar heating, which results in thunderstorms (29,30).

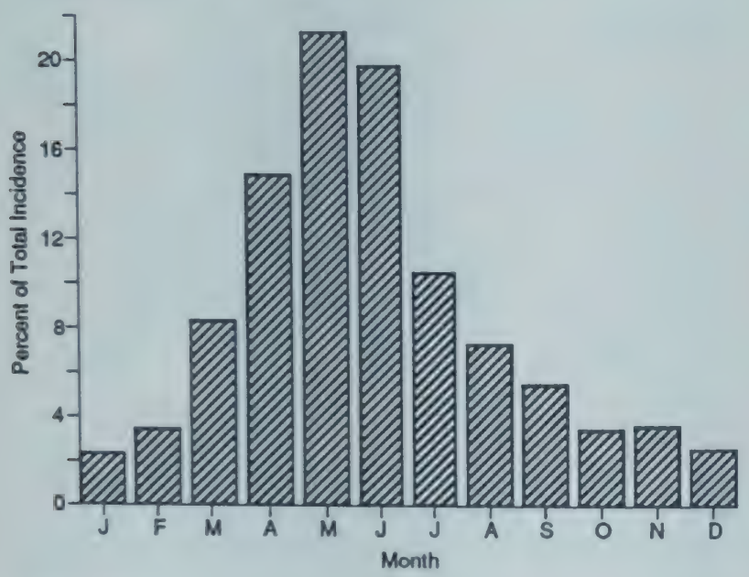
The percentage distribution of tornado incidence by month is shown in Figure 4. Months of greatest tornado incidence were May (21%) and June (20%). By April 28, 25%

FIGURE 3. Percentage distribution of tornadoes, by time of day, United States, 1950-1970



Source: Reference 17.

FIGURE 4. Percentage distribution of tornadoes, by month, United States, 1950-1970



Source: Reference 17.

of the tornadoes had occurred; June 4 represented the 50% point; and 75% were reported by July 20. Only one date, January 16, had no reported tornadoes. Data pertaining to the 1982 monthly incidence of tornadoes, killer tornadoes, and tornado-associated fatalities are shown in Table 3. Again, May and June accounted for the largest percentages of tornado incidence. However, at least for this 1-year period, the largest percentages of lethal tornadoes and tornado-associated fatalities occurred in April.

For public health risk, not only is the number of tornadoes that have occurred in a particular area over a specified time important, but also the sizes of the storm and associated tornadoes (17). Approximately one-half of all tornado-associated fatalities between 1965 and 1974 resulted from only two tornado storms (21). Two hundred seventy-one persons were killed on April 11, 1965, when a series of tornadoes struck the Midwest. On April 3, 1974, a series of more than 100 tornadoes killed 318 persons in 11 states in the South and Midwest. Also, the surface area damaged by a tornado is important (31-33). Extensive analysis of path areas has shown an association with region; mean path areas include 2.82 square miles for Iowa (31), 1.82 square miles for 13 eastern states (29), and 0.96 square miles for all states east of the Rocky Mountains (33). The tornadoes with longer tracks may be more lethal and damaging than those with shorter tracks (34). The characteristic longer path lengths of lethal tornadoes may result from a correlation between path length and intensity (35). Analysis of 14,409 tornadoes in the United States showed that just over 2% were violent (F4 or F5). However, these 340 violent tornadoes caused 68% of the fatalities attributed to tornadoes (9). In the 1966 tornado in Topeka, Kansas, all mortality occurred directly within the path area, while serious morbidity occurred within a broader area (36).

On March 29, 1984, 22 tornadoes covered a 300-mile area through North and South Carolina, resulting in 57 deaths, thousands of people injured, and more than $200 million in damages (22). Kindel contends that building practices, not tornadoes themselves, were largely responsible for the damage. Buildings of unreinforced masonry and large areas of glass are likely to suffer the most extensive damage. He submits that if one considers the costs of deaths and damage, it is economically feasible to design buildings that will resist tornado damage.

However, this issue may not be so clearcut; it may not be so easy to generalize about how the type of permanent housing is related to tornado casualties. Frame and masonry structures appear to have separate collapse models (28). Frame homes have more ventilation and greater possibility for pressure equalization. They are more likely to have roofs ripped off first and then have the walls strewn along the tornado path. In contrast, walls of masonry buildings push outward in response to the internal force caused by the pressure reduction outside, causing the roof to fall in. However, a big difference may result from reinforcing the masonry so that it will not move outward.

TABLE 3. Monthly distribution of tornadoes, killer tornadoes, and tornado fatalities, United States, 1982

	Tornadoes		Killer tornadoes		Fatalities	
Month	Number	(%)	Number	(%)	Number	(%)
January	18	(1.8)	1	(3.1)	1	(1.6)
February	3	(0.3)	0	(0.0)	0	(0.0)
March	60	(5.8)	6	(18.6)	6	(9.4)
April	150	(14.6)	10	(31.2)	30	(46.9)
May	329	(32.0)	4	(12.5)	14	(21.9)
June	196	(19.1)	4	(12.5)	4	(6.2)
July	95	(9.3)	0	(0.0)	0	(0.0)
August	34	(3.3)	0	(0.0)	0	(0.0)
September	38	(3.7)	2	(6.3)	2	(3.1)
October	9	(0.9)	0	(0.0)	0	(0.0)
November	24	(2.3)	0	(0.0)	0	(0.0)
December	71	(6.9)	5	(15.7)	7	(10.9)
TOTAL	1,027		32		64	

Source: Reference 10.

Human

Age is an often reported characteristic of persons who sustain tornado-associated injuries. However, most statistics on age appear to come from reports concerning individual case histories or aggregate case series. For example, a record review of all patients (24) treated at one major medical center as a result of a 1982 Arkansas tornado showed that ages of patients ranged from 5 to 69 years, with a mean of 35.5 years (2). Characteristically, information on the age distribution of the population at risk was not provided.

However, two published studies do provide examples of interpreting patient-age distributions relative to those of the exposed population. With reference to the 1979 Wichita Falls tornado, rates of fatal and serious injury increased with age (19). Persons > 60 years of age were seven times more likely to be injured than individuals < 20 years.

On the evening of March 28, 1984, a series of tornadoes in North Carolina and South Carolina resulted in over 1,000 casualties (people killed or injured) and $100 million in property damage (23). Residents of the affected North Carolina counties ≥ 55 years of age made up 18% of the population. Of those in North Carolina with nonfatal injuries and recorded ages, 106 (16%) of 660 were in this age group; of the persons killed, 19 (43%) of 44 were in this group ($p < 0.0001$).

Any apparent increased risk of injury associated with elderly populations may be the consequence of numerous factors, including greater susceptibility to injury from comparable amounts of mechanical energy, location at time of tornado touchdown, medical conditions that might adversely affect comprehension of or reaction to warnings, and living in isolation.

Another postulated risk factor is gender. The assessment of its contributions has limitations similar to those just discussed for age: a preponderance of numerator data not suitable for determining risk. For example, the medical record review of 24 tornado victims in Arkansas showed that 17 (71%) were males and seven (29%) were females (2). In contrast, analysis of population-based data from the 1979 Wichita Falls tornado study showed that above the age of 40 years, women were at greater risk of injury than men, and above 60 years, this risk was approximately twice as great (19). Concerning this observed sex differential, Glass et al. hypothesized that the greater occurrence of osteoporosis among women made them more susceptible to sustaining fractures. However, differences in location and responsibility for others might also be factors.

The best estimates of risk associated with an individual's location come from the Wichita Falls tornado study (19). Compared with persons located in permanent, single-family houses, occupants of mobile homes were approximately 40 times more likely to sustain a serious or fatal injury, and occupants of automobiles were at approximately five times greater risk. One limitation of these data was the small number of cases (four) that occurred in mobile homes. However, regardless of the exact magnitude of risk differential, it is clear that the most dangerous place to be when a tornado strikes is in a mobile home. Detailed analysis of the injured occupants of automobiles provided an interesting finding. Persons in automobiles accounted for 26 (60%) of the 43 traumatic deaths and 30 (51%) of the 59 serious injuries. Of these 56 individuals, 43 (77%) had entered their cars with the deliberate intention of outrunning the tornado.

Results from the 1984 Carolinas tornado disaster study further substantiated the increased risk associated with mobile homes (23). Emergency room records listed the location of each person at the time of injury for 346 (39%) of the 896 nonfatal injuries. Death certificates recorded this for 55 (93%) of 59 persons killed. When location was recorded, 19 (46%) of 41 North Carolina fatalities and 176 (51%) of 348 injured persons in North Carolina and South Carolina were in trailers or mobile homes at the time of injury. In the counties that sustained tornado damage, an estimated 11% of residents lived in trailers or mobile homes.

The vulnerability of a person in a motor vehicle may be related to the vehicle's susceptibility to being lifted. During a 1965 Indiana tornado in all cases in which auto occupants were injured or killed, the vehicle was completely raised off the ground (37).

Review of published literature showed trends in the characteristics of injuries (fatal and nonfatal) sustained by victims of tornado disasters. The leading causes of death are craniocerebral trauma followed by crushing wounds of the chest and trunk (38). As a result of a 1965 Indiana tornado, 17 persons were killed immediately (37). Fourteen died of craniocerebral trauma, two had crushed-chest trauma, and one had cervical spine fracture with cord injury. One other injured victim died while in the emergency room of a local hospital. Of the 44 injury deaths resulting from the 1979 Wichita Falls tornado, 43 persons died of multiple trauma within minutes of the tornado touchdown. The other person died within 24 hours.

Fractures are the most frequent type of nonfatal injury. They accounted for 57% of all morbidity in the Worchester tornado, 66% in the Dallas tornado, and 86% in the Wichita Falls tornado. Lacerations and other soft-tissue injuries are also frequent. In some tornado disasters these injuries have resulted in contaminated wounds (37,39).

In the 1984 Carolinas tornado disaster study (23), medical records for 874 persons contained adequate information. Of this group, 331 persons (38%) sustained one or more fractures, 496 (57%) sustained one or more lacerations, and 432 (49%) sustained one or more contusions.

After the 1965 Indiana tornado, 24 patients were admitted to the hospital; all had multiple injuries caused by objects moving at high velocities (37). Percentage of patients with specific injuries included soft tissue trauma (100%), head injury (60%), shock (50%), skeletal fractures (43%), and kidney trauma (13%). Fracture incidence by percentage of patients included head (25%), upper extremities (16%), thorax (8%), vertebrae (8%), pelvis (8%), and lower extremities (35%).

It is important to know whether exposed populations received any official warning about approaching tornadoes. Interviews with 50 victims of the 1953 Worchester tornado disaster showed that no one had any official warning (40). After the 1979 Wichita Falls tornado (19), 96% of all people surveyed felt that they had been adequately warned that the tornado was approaching: sirens had sounded twice beginning 1 hour before the tornado struck, and all local radio and news stations were reporting the National Weather Service advisories. For the tornado outbreak on April 3-4, 1974, 28 tornado watches were issued, and within these tornado-watch areas, 285 of the 315 deaths occurred (5). Analysis of

14,600 tornadoes occurring between 1952 and 1973 showed that of the 2,575 fatalities, 66% occurred within declared tornado watch areas (24). For these deaths the warnings may not have been heeded, or the tornadoes may have been more severe than expected.

When a tornado warning is issued, persons within the potential path of the storm should take immediate protective measures. Interviews with 50 victims of the 1953 Worchester tornado disaster indicated that no one recognized hailstones to be a tornado indicator (they actually placed themselves at additional risk by going outside to look at the hail); of the 22 who saw the approaching tornado, only 14 recognized it as a tornado; and, only eight took measures to protect themselves (40). As a result of the 1979 Wichita Falls tornado, 59 people were injured seriously enough to be hospitalized for a week or longer. Physicians commented that most patients who received major abrasions and lacerations had not covered themselves with blankets, pillows, or mattresses (19). Data pertaining to 24 patients injured by a 1982 Arkansas tornado showed that only two seriously attempted to prevent injury: one man hid behind a couch, and one woman lay face down in the bathtub with a pillow over her head (2).

One published study has examined "personality" as a risk factor (28). This assessment of a disproportionately higher frequency of tornado-related deaths in the South compared with the North examined such factors as frequency of tornado occurrence, tornado severity, diurnal distribution of tornadoes, population density, and structural integrity of housing. The researchers concluded that no important differences in these factors could have impacted on the mortality differential. Interviews were conducted with 57 respondents, 33 from Illinois and 24 from Alabama. All were white women between the ages of 31 and 60 who had at least an elementary school education. Results of a sentence-completion test led the authors to conclude that Southerners, in contrast to Northerners, were more likely to consider a tornado to be an act of God and less likely to try to take control over their fate. They were more likely to trust "luck," to be less responsive to weather warnings, and to have a passive and fatalistic attitude. However, the authors did not provide any information about how subjects were selected, the number who refused, or how representative they were. The results of this study should be interpreted cautiously until they are replicated since they are based on a small behavior sampling of a small number of respondents.

It has been widely believed that natural disasters have a uniformly adverse effect on the emotional health of their victims. It has been hypothesized that the experiences sustained during and after a disaster may cause victims to develop severe psychopathology or disaster syndrome (unrealistic absence of emotion along with docility, indecisiveness, and trancelike behavior). It appears that, at least for tornado disasters, any belief about increased significant mental health problems is supported more by supposition than by scientific data (41,42).

Several published studies have found no association between experiencing a tornado disaster and developing serious psychopathology. Interviews with 111 persons 5 months after a tornado struck Omaha, Nebraska, in 1975 showed neither serious psychopathology nor social disorganization (41). This study showed that residents of neighborhoods hit by a tornado readily recovered from the emotional stresses.

These findings are similar to those from an evaluation of another tornado disaster; no coping impairments were observed among victims in Topeka, Kansas (43). Interviews with 26 victims 5 months after a tornado struck Joplin, Missouri, in 1973 showed that the disaster did not produce severe emotional impairment and few victims felt the need for professional mental health assistance (44). Most persons interviewed believed that the emotional reactions were natural and would pass in time, especially as financial problems were resolved and as they experienced less-destructive storms. Surveys of victims of the 1974 Xenia, Ohio, tornado were conducted 6 months and 1 year after the disaster (45). These victims did not report any pathologic or severe psychological behavior. In fact, 27% of the interviewees stated that relationships with close friends and family had improved.

The literature does contain opposite contentions about this association. However, it is important to examine the case definitions used in such studies. For example, in a study of tornadoes that struck Waco and San Angelo, Texas, in 1953, selected families were interviewed 4 months or longer after the disaster. Results showed that 17% of the black and 30% of the white informants said that a family member had undue fear of the weather (46). Interviews with 142 victims of the tornado that struck Dallas, Texas, in 1957 showed that 72% of the victims believed that a member of their family had become nervous or upset since the tornado (47). However, it is extremely important to distinguish between psychological apprehension (or stress) and severe mental illness. The former is to be expected to some degree and may have little true impact on public health. The latter appears to be the true public health concern.

Results from studies of other types of disasters suggest that factors that may influence the incidence of mental illness include vulnerability of individuals, the severity of the disaster, and the strength of the support systems for the affected neighborhood. With respect to individual vulnerability, one potential risk factor is age. Young children and the aged may be more adversely affected. Also, limited coping skills and histories of mental illness may be important. Consequently, populations with large percentages of very young, elderly, or marginally adjusted people may be at higher risk.

The severity of the disaster also affects the ability of individuals to cope. Degree of adjustment is based on the duration, nature, and extent of the changes in living situations produced by the disasters. Studies have shown that the non-tornado-associated natural disasters that caused the largest widespread impairments in personal functioning produced the longest and most extensive disruption of living situations (48,49). Also, the immediate and long-term support systems available to disaster victims affect their capability to cope effectively (50).

Only the study of the flash flood resulting from the Buffalo Creek Dam collapse in West Virginia showed severe mental illness among most victims. Researchers found that 93% of the survivors had major psychiatric disturbances. But this was a very unusual disaster. Almost every family lost at least one member, and the entire settlement was physically destroyed along with the social and cultural systems (45).

Many of the studies to evaluate the association between tornado disasters and severe mental illness appear to have limitations pertaining to subject selection, study design, and hypothesis selection. The number of subjects is often small, with no information provided about participation and representativeness. Studies often focus upon a fixed point in time after the disaster and totally depend on self-diagnosis of illness and need for mental health care. Hypotheses usually examine a subjective outcome with little assessment of relative importance of impact on normal daily activities or outlook.

Public Health Prevention Strategies

Epidemiology: Surveillance and Research

Epidemiology has the potential for playing a key role in minimizing the public health impact of tornado disasters. With other public health problems, epidemiology is used to complement activities of and conclusions reached by other scientific disciplines. The same need exists for evaluating and preventing tornado-associated morbidity and mortality. To maximize the efficacy of prevention strategies, the relative role of epidemiology may need to be increased.

To date, the lead role in public health prevention appears to have been assumed by physical scientists (meteorologists and structural engineers), disaster-relief planners, and health-care providers. Epidemiologic consideration and assessment have been limited to a handful of studies that, compared with applications to the study of acute and chronic diseases, are very basic and more descriptive than analytic. The full spectrum of epidemiologic activities encompassing both surveillance and research appears to be an appropriate strategy for public health prevention.

Traditional surveillance activities could be used for two different objectives. First, surveillance could generate population-based descriptive statistics and crude assessments of risk. Second, surveillance could help assess trends in tornadoes and associated mortality/morbidity and assist in determining the general efficacy of specific prevention and control measures.

Epidemiologic research, consisting of descriptive and analytical studies, could provide new information on characteristics and risk factors and possibly attempt to replicate existing findings. Replication appears necessary in some cases because of limitations in how the information was collected or analyzed. Especially important is the *a priori* selection of hypotheses and protocols.

Prevention and Control Measures

A tornado is the most difficult weather phenomenon to forecast precisely (21). By detecting weather conditions that usually precede the generation of tornadoes (i.e., thunderstorms), it is possible to identify geographic areas with high probabilities. This forecasting is done by the National Severe Storms Forecast Center in Kansas City, Missouri. This Center, in conjunction with local National Weather Service (NWS) offices, has a sophisticated tornado-watch and tornado-alert system to provide as much warning as possible to the public.

The total warning system for tornadoes is based on three components: observations and analyses, forecast preparation, and warning preparation. Observations come from NWS offices, Department of Defense installations, observer networks, radar stations, and satellite stations. Forecast preparation is done by the National Severe Storms Forecast Center in Kansas City. Tornado watches and warnings are prepared and issued by the Weather Service Forecast and NWS offices, each with designated counties of responsibility. Because is not possible to predict exact times or places of occurrence, areas 100 miles wide by 250 miles long can be identified that have weather conditions suggesting tornado generation. Tornado watches address these high-probability areas by indicating the area affected and the period of time during which the probability of a tornado is high. Watches are sent to the local offices of the NWS and disseminated to the population affected via radio and television announcements. Tornado warnings are issued when a tornado has been either sighted or indicated by radar. Warnings indicate the location of the tornado at the time of detection, the area through which it is expected to move, and the time period during which the tornado will move through the area warned. When a tornado warning is issued, persons in the path of the storm should take immediate safety precautions.

On March 21-22, 1952, an outbreak of 28 tornadoes near Dierks, Arkansas, resulted in 204 deaths and $15 million in property damage (5). This tornado outbreak involved the first tornado watch issued by the NWS. Several factors affected the efficacy of this watch. First, community warning plans and systems had not been fully developed. Second, information dissemination was slow and inadequate for effectively conveying warnings. Third, the education and cooperation of appropriate agencies and the population at risk were insufficient.

To ensure the maximum efficacy of tornado watches and warnings, it appears essential to have local implementation. This activity is best ensured by establishing and using community-action networks. These networks would have numerous functions that could directly impact the public health efforts associated with the event phase of a tornado disaster. First, a warning center along with an observer network covering a 20-mile radius should be established. Second, the network should receive NWS watches and warnings along with reports from observers in the field. Third, the network should notify the local NWS office of any sightings and should notify endangered towns of all sightings and warnings. Fourth, a system of positive alerts (fire alarms, civil defense sirens) should be established. Five, awareness of these activities should be maintained through rehearsals and public reminders. Sixth, the network should encourage and help support the education of community residents about risks associated with tornadoes and appropriate actions for minimizing these risks.

The importance of local warning systems is perhaps best illustrated by example. At 4:29 p.m. on May 6, 1975, a tornado hit Omaha, Nebraska (51). This tornado was 600 yards wide, had winds up to 200 mph, and traveled along the ground for about 9 miles. In the 200-block area directly affected by the tornado, 31,000 people lived or worked. Three persons were killed and 200 injured. The staff of the National Oceanic and Atmospheric Administration believed that had it not been

for the civil defense sirens and television and radio warnings, 500 or more people would have been killed.

The importance of a tornado disaster-preparedness program was assessed by comparing the effects of two tornadoes that hit the same town: one before the program was established and the other after (52). In 1968 when the first tornado hit, the community of Jonesboro, Arkansas, had a population of 25,000 people. The town had no community warning center, no observer network, no television or radio dissemination of watches and warnings, no educational programs, and no disaster-drill procedures. The tornado resulted in 34 deaths, 82 hospitalizations, 458 injuries, and $8 million in property damage. When the second tornado hit in 1973, the community of 29,000 had implemented and was conducting all the previously mentioned activities. This tornado resulted in two deaths, 21 hospitalizations, 246 injuries, and $50 million in property damage. This drastic reduction in public health impact—even though this tornado was apparently of greater strength—certainly supports the efficacy of such preventive activities.

It is important to realize that regardless of the efficiency of national and local information-dissemination networks, it is essential that the population at immediate risk from a tornado has access to adequate protective shelter. On May 22, 1987, a multiple-vortex tornado with winds of up to 260 mph struck Saragosa, Texas. This tornado resulted in 30 deaths and 131 injuries (53). Most of these deaths and injuries occurred within a community hall where a graduation ceremony for preschool children was being held. Neither the community nor the county had a pre-identified tornado shelter, and the community hall could have been the safest structure available. Most buildings could not withstand high winds and were constructed of adobe or concrete blocks. The tornado-associated incidence of injury was high (approximately 70% of the persons located within the swath of the tornado were injured), probably due to the lack of protective shelter.

Disaster contingency planning for tornadoes outlines specific actions to be undertaken after tornadoes have struck. Federal and state agencies have plans that address physical needs of the victims and clean-up of affected areas. With respect to specific public health considerations, the literature seems to focus on recommended actions for health-care providers (36,54-57). Many articles discuss the involvement of hospitals or physicians immediately after tornado disasters. For example, the 1979 Wichita Falls tornado disaster resulted in the treatment of 1,000 people at local hospitals and the hospitalization of 130. Just 6 days before the tornadoes struck, the hospitals participated in a citywide civil defense emergency drill. The article describing this event concluded that this practice greatly contributed to the ability to handle and treat patients (58).

Most articles provide personal accounts of health-care delivery. Overall, these subjective evaluations are extremely positive about the ability to effectively handle case loads and the medical problems encountered. The most important factors in this success appear to be prior development and rehearsal of a disaster plan. Common components of plans that frequently need to be emphasized or improved include keeping detailed, accurate, and easily retrievable medical records; having sufficient back-up emergency electricity; controlling visitors and relatives; and offering a program of adequate radio messages.

Strategies pertaining to the prediction of, warning about, and planning for tornadoes cannot be totally successful without the dissemination of appropriate information to educate persons frequently exposed to tornadoes. Such persons should have a sufficient understanding of both tornado etiology and characteristics of preventive actions. This understanding is especially important for persons (such as parents, teachers, hospital and nursing home employees) who are responsible for others. Unfortunately, the literature cites numerous instances in which tornado victims have inadequate knowledge. Examples include not recognizing a tornado when it is spotted, going outside to look at hailstones from thunderstorms, not understanding the difference between a tornado watch and warning, and making no serious attempts to prevent injury. These examples and others demonstrate a tremendous need for increased education through dissemination of existing information.

The National Oceanic and Atmospheric Administrations disaster preparedness staff has current preventive guidelines, which they recommend persons become familiar with and use in case of a tornado. These guidelines are based on the location of the person at the time a tornado strikes. People in permanent homes should seek refuge in a basement, hallway, closet, or interior room and cover themselves with pillows, blankets, or mattresses. Persons in mobile homes are urged to seek a more substantial structure or shelter. Proper tiedowns are totally ineffective when windspeeds exceed 50 mph. Persons driving cars in rural areas should drive in a direction perpendicular to the tornado's path; if this is not possible, they should leave their vehicle and lie flat in the nearest ditch or gully. Persons driving in urban areas should leave their vehicle and seek shelter. In high-rise buildings, people should seek shelter in small interior rooms or hallways.

Increased educational efforts should include two considerations. First, existing prevention guidelines are based on structural-damage assessments primarily, and, to a lesser degree, on actual-risk assessments. The guidelines represent what are perceived to be the safest actions to take. However, people can always cite exceptions. For example, during a tornado outbreak in Illinois on April 11, 1965, a family of three followed recommended safety practice by seeking refuge in their basement (5). All three were killed when they were literally blown out of the basement of their leveled home and carried two blocks. It is important to stress that on the basis of current knowledge, these guidelines maximize but do not guarantee the safety of persons exposed to tornadoes. Second, the frequent occurrence of tornadoes and tornado watches in many parts of the country may desensitize persons to the level of risk. In many areas of the United States, the broadcast of tornado watches and warnings is common during the spring. Many people may become lackadaisical about their response (28). However, this type of response would certainly appear to increase potential risk of injury. Educational activities should attempt to maximize awareness and encourage appropriate response.

Critical Knowledge Gaps

The literature pertaining to public health impacts of tornado disasters is fairly extensive and varied. Excluding con-

siderations of research quality, published reports have indicated that mortality and morbidity from tornadoes may result from a number of factors, including the strength and path area of the tornado, population magnitude and density, time of day, type of building, storm-warning procedures, public education and acceptance of storm-warning procedures, and safety precautions taken. Overall, the quality and quantity of research studies designed specifically for the evaluation or prevention of public health impacts are extremely limited. Several critical gaps in knowledge must be addressed if substantial improvements in prevention are to be made in the future.

Documentation of total public health impact has limitations with respect to purpose, comparability, and detail. Data on mortality and morbidity are generated and used by different agencies (NWS, Federal Emergency Management Agency, American Red Cross) for different purposes. Also, the criteria and methodology for collecting this information vary. The data collection and reporting are done by agencies whose primary objectives are neither public health research nor practice. Information on the comparability and validity of these data is needed so that specific recommendations can be made for standardizing them.

Existing work seems to overemphasize on mortality at the expense of morbidity. Although premature and unnecessary mortality is certainly a part of the public health problem associated with tornadoes, nonfatal injuries and the need for emergency care constitute the major portion of the problem. More detailed information is needed about the characteristics of tornado disasters that cause such outcomes, the circumstances associated with their occurrence, and their impact on daily activities and quality of life.

For proper public health concern, there appears to be undue emphasis on "tornadoes" rather than on "tornado disasters." Data presented earlier in this chapter clearly show that only a percentage of all tornadoes was responsible for most of the public health impact. These tornadoes are usually the more violent ones associated with large tornado storms. More emphasis should be placed on these tornado disasters and less concern given to tornadoes in general, many of which have no public health impact.

Although various factors have been associated with adverse public health outcomes, there is insufficient information about the concomitant contributions of these factors. Currently, the only method for ranking their importance is conjecture. Information based on results of multivariate analyses of epidemiologic data would be helpful for determining the relative importance of various factors and priorities for research. For example, authors have concluded that the trend of decreasing fatalities and increasing numbers of reported tornadoes is indicative of the success of tornado warning systems (21). However, the scientific data supporting this cause and effect relationship apparently do not exist. More specific information is needed for high-risk areas of smaller land size. Most geographic information pertains to states or regions. In consideration of discussed control strategies (community action networks), information is needed at the county level.

For high-risk areas, more validly documented information on mortality and morbidity is needed. Several studies have shown problems with the completeness of records kept by hospitals, physicians, and vital statistics personnel. Knowledge about the specific problems and solutions to these problems is needed. Records should not only be more complete but also include more descriptive information.

Current tornado warning systems appear to be well organized. However, extensive mortality and morbidity have been reported within areas covered by tornado watches and warnings. Additional information is needed about dissemination of this information at local levels. For example, little information appears to be available about the presence and characteristics of community-action networks within these areas. Also, information is needed about the comparative effectiveness of radio and television broadcasts versus positive alerts (sirens). In the warning process, the relationship between the time of day a tornado occurs and the radio and television habits of the population at risk is very important. During the afternoon when audiences are smallest, use of positive alerts is most important (27).

Research Recommendations

To enhance the efficacy of strategies designed to prevent or mitigate public health impacts of tornadoes, the following research activities are recommended.

1. Future efforts should emphasize descriptive and analytical epidemiologic studies, as necessary. Emphasis on case-reports or case-series data should be minimized.

2. The feasibility of adding pertinent epidemiologic components to existing tornado contingency plans used by Federal and state agencies should be evaluated.

3. Collaborative efforts among agencies dealing with tornadoes should attempt to systematize case definitions and data collection to maximize the usefulness of public health data routinely collected and analyzed.

4. Surveillance efforts need to be improved by including more detailed public health indices, revising International Classification of Diseases mortality classification codes so that they are specific to tornadoes, and encompassing more detailed morbidity and emergency-care information.

5. Surveillance efforts should identify high-risk areas, preferably at the county level. When feasible, county-specific maps for high-risk states should be generated from race- and sex-specific, age-adjusted mortality and morbidity rates.

6. Criteria for basic data elements should be developed for medical and legal records used for tornado victims in high-risk areas.

7. Numerous epidemiologic surveys should be conducted in high-risk areas and focus on such issues as presence and characteristics of community-action networks, number of trailer parks with and without underground shelters, and public knowledge of tornadoes and preventive actions.

8. Epidemiologic studies should be conducted to evaluate the contributions of warning systems, public education,

and health-care delivery to minimizing public health impacts from tornado disasters.

9. A case-control study is needed to provide data suitable for multivariate analysis of risk factors so that the relative importance of individual factors can be assessed.

10. The need for long-term studies that better quantify tornado-associated emotional problems and their impact on the well-being of victims should be assessed.

Summary

Tornadoes are the most lethal and the most violent of all natural atmospheric phenomena. Although almost 700 tornadoes strike in the United States each year, only about 3% are violent enough to result in public health disasters; these tornadoes are usually part of tornado storms. In the United States from 1970 to 1980, tornadoes resulted in death for 856 persons, injury for 22,012 persons, and emergency-care requirements (shelter, clothing, food, or medical supplies) for 909,605 persons.

Published literature from several scientific disciplines identifies various factors that may influence public health outcomes associated with tornadoes. Environmental factors include geographic location, time of day and month of year, size and number of tornadoes, and building practices. Human factors include age, sex, location, type of warning, and protective measures.

Appropriate public health prevention strategies appear to consist of three broad categories of activities: epidemiologic surveillance and research; control measures involving forecasting and warning, local implementation, and contingency planning; and dissemination of information to and education of the public at risk.

Additional epidemiologic activities in this field are needed if further reductions in tornado-associated mortality and morbidity and improvements in emergency care are to be made. Specific critical knowledge gaps and research recommendations are outlined.

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Heat Waves

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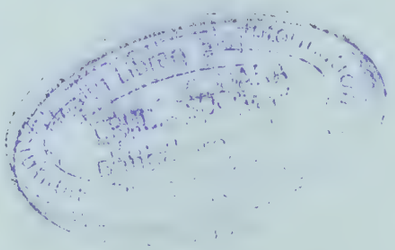


Background and Nature of Physical and Physiologic Aspects of Heat Exposure

Air temperature, humidity, air motion, and radiant heat energy are the most important factors for persons to have heat stress. Of these, air temperature is the most important. When the air temperature is low, metabolically generated heat is easily lost from the body to the air by convection. As air temperature increases, convective heat loss occurs less readily until, at temperatures above body temperature, convective heat loss is no longer possible, and heat may be gained from the air. High humidity limits the cooling effect of the evaporation of perspiration, and therefore leads to increased heat stress. Increased air speed facilitates convective heat transfer and the evaporation of sweat. Radiant-heat energy adds to heat stress, independent of the other three variables. For example, radiant heat causes one to feel hotter in direct sunlight than in the shade, even under identical air temperature, humidity, and air speed (1-3).

Several indices of heat stress have been developed that may be useful to the public health professional in assessing how much heat stress could result from a given set of environmental conditions. Such indices yield one number designed to reflect the combined effects of some or all of the four environmental variables described above. The purpose is to give a single figure describing how hot "it feels" to a person exposed to specific environmental conditions (4). Two such indices are the "Effective Temperature," an empirically derived value, and the "Apparent Temperature," derived mathematically using physical and physiological principles (1-6). Other useful indices of heat stress also exist. A complete discussion is beyond the scope of this chapter, but the subject has been well reviewed elsewhere (4).

Heat-stress indices have limitations. Most indices involve implicit assumptions about metabolic heat production, clothing, and body habitus. Since these parameters vary among people, the predicted value of heat stress for any single person is, at best, an approximation. Certain heat indices are difficult to use because the raw data required for their calculation are not easily available. Finally, it should be recognized that an index level using meteorologic observations at a weather station may differ from the level that would be obtained if one could measure the microclimates to which individuals are subjected.

Health Effects of the Heat

The Spectrum of Heat-Related Illness

The illnesses directly attributable to prolonged periods of high environmental temperature are heatstroke, heat exhaustion, heat syncope, and heat cramps. Heat waves may also increase morbidity and mortality due to other illnesses that can occur even in the absence of heat stress. Burns, which result from the local application of intense heat, are not considered here.

Heatstroke occurs when perspiration and the vasomotor, hemodynamic, and adaptive behavioral responses to a heat stress are insufficient to prevent a substantial rise in core body temperature. Although standardized diagnostic criteria do not exist, a patient's condition is usually designated as heatstroke when rectal temperature rises to ≥ 105 F (40.6 C) as a result of high environmental temperatures. Mental status is affected, and the patient may be delirious, stuporous, or comatose. Classically, sweating is said to be absent in heatstroke, but this is not always so.

Heatstroke is a medical emergency. Rapid cooling—usually by means of ice massage, ice-water bath, or special facilities for evaporative cooling—is essential to prevent permanent neurological damage or death. Further treatment is supportive, and admission to an intensive-care unit is often required. The outcome is often fatal, even with expert care. The death-to-case ratio in reported case series varied from 0 to about 40% and averaged about 15% (7-17).

Heat exhaustion is a much less severe disease than heatstroke. Patients may complain of dizziness, weakness, or fatigue. Body temperature may be normal or slightly to moderately elevated. The cause of heat exhaustion seems to be fluid and electrolyte imbalance due to increased perspiration in response to intense heat. Therefore, treatment is directed toward the normalization of fluid and electrolyte status, and the prognosis is generally good (13).

Heat syncope refers to the sudden loss of consciousness, usually associated with exercise, by persons who are not acclimatized to hot weather. Consciousness returns promptly with assumption of a recumbent posture. The cause is thought to be circulatory instability due to superficial vasodilation in response to the heat, and the disorder is benign (18). Heat cramps occur during exercise done by persons unaccustomed to the heat. The cramps are thought to be due to mild fluid and electrolyte imbalances and generally cease to be a problem after acclimatization (13).

Heat-Wave-Associated Mortality

Currently in the United States, in years during which no major heat wave occurs, an average of approximately 200 deaths are recorded on death certificates as having been caused by the heat (19). A few such deaths occur during winter and the cooler months of the year, indicating that not all of them are caused directly by meteorologic conditions, but the great majority occur during summer. In years in which prolonged periods of abnormally high temperatures (heat waves) affect large areas of the country, the number of deaths attributed to heat rises greatly. In 1980, when summer temperatures reached all-time high levels in much of the central and southern United States, over 1,700 deaths were diagnosed as heat-related, over seven times the number expected if there had been no heat wave (20).

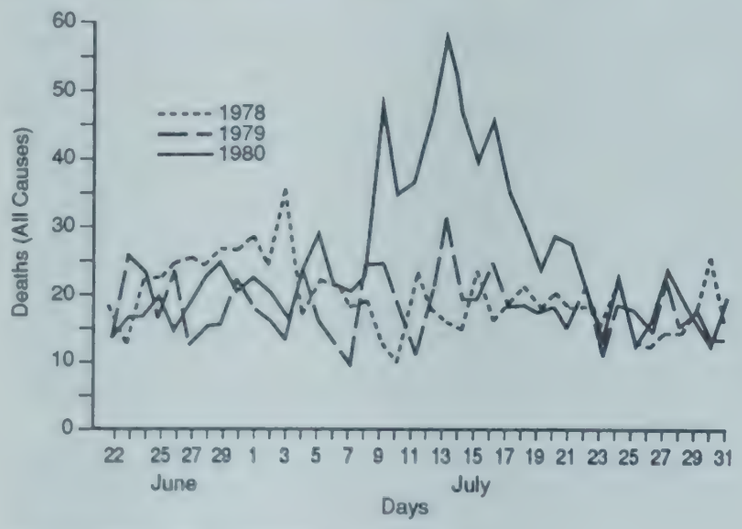
Such figures, however, do not reflect the full extent of the problem. In previous studies only a portion of the increase in mortality during heat waves has been documented on death certificates as having been caused by the heat (21). Diagnoses of heat-related death have regularly underestimated heat-wave-associated excess mortality* by from 22% to 100% (Table 1) (22-28). Moreover, mortality figures give no indication of the substantial amount of non-fatal illness that occurs as a result of the heat.

Of the syndromes whose sole cause is environmental heat, heatstroke is the only one with a substantial death-to-case ratio. Thus, it is reasonable to suppose that the great majority of deaths diagnosed as caused by heat represent mortality due to heatstroke. This supposition has been checked only rarely, but existing data tend to support it. Henschel and others reviewed the hospital charts of 120 persons whose deaths had been certified as heat related and found that virtually all had a temperature of ≥ 103 F (39.4 C) upon hospital admission, a fact which they interpreted as showing that most of these deaths were due to heatstroke (28). In another series only 60% of 57 persons hospitalized for physician-diagnosed heat-related illness and who later died met the author's definition of heatstroke. However, the criteria used for diagnosing heatstroke were very strict (25). Current death-certificate coding practices at the national level in the United States make it difficult to evaluate the precise clinical diagnoses leading to heat-related death since, because deaths are coded by their external cause, environmental heat (*International Classification of Diseases, 9th Revision, Code E900*), rather than by the specific illness that the heat produces.

Mortality associated with a heat wave is often so great that it appears as a sudden and substantial increase in the total number of deaths occurring in a given area (Figure 1). Increases of over 50% in the crude mortality rate are not uncommon. Moreover, despite the increased use of air conditioning, there has been no clear and substantial decrease in the death toll taken by heat waves in recent years (Table 1).

Stroke (brain infarction or hemorrhage) was found to be an important cause of heat-wave-associated death by Schuman and others, who found that deaths due to "cerebrovascular accident" rose 82% and accounted for 52% of the excess mortality caused by a heat wave in Detroit, Michigan, in 1963. During another Detroit heat wave in May of the previous year, they observed a similar rise in deaths from stroke of 104% (26% of all heat-wave-associated deaths) (23). When Schuman studied the July 1966 heat wave in New York City, he found a far less dramatic increase of 27%, accounting for a little over 6% of an estimated 1,181 excess deaths caused by the heat. He felt, however, that the coding of stroke deaths in New York was different from that of other cities and tended to underestimate the problem (29).

FIGURE 1. Trends in daily deaths showing the increase in numbers of deaths associated with the July 1980 heat wave, St. Louis, June 22-July 31, 1978-1980



Source: Missouri death certificates.

Other investigators have observed that certified stroke deaths increase during severe heat (Table 2). However, the magnitude of the increase has generally been less than that noted in the Detroit studies. Increases have ranged from 25% to 55%, accounting for about 5% to 20% of heat-wave-associated mortality (24,30). An increase in hospital admissions for persons with non-fatal strokes has also been reported (31).

The variability in the magnitude of the increase in stroke mortality relative to other causes of death that apparently increase during the heat suggests that some deaths due to causes other than stroke are misclassified as stroke and/or that some stroke deaths are misclassified as being due to other causes. However, there is evidence for increased coagulability of blood in heat-stressed persons, and such increased coagulability may be the biological basis for an

*In this chapter "excess mortality" or "excess deaths" during a heat wave refer to the difference between the number of deaths actually observed during some specific period of hot weather and the number of deaths expected based on the crude death rate in the same geographic area during some appropriate control period which neither a heat wave nor any epidemic was present.

TABLE 1. Numbers of total deaths observed, total deaths expected, excess deaths, and deaths attributed to the heat in specific locations during selected heat waves, United States, 1872-1980

Location/period	Reference	Observed deaths	"Expected" deaths	Excess deaths	Excess deaths (as a percentage of expected)	Deaths classified as related to heat	Deaths related to heat (as a percentage of excess deaths)
Birmingham, England							
June 24-July 8, 1976	22	491	384*	107	27.9	0	0.0
Detroit, Michigan							
May 12-May 18, 1962	23	429	325†	104	32.0	0	0.0
June 23-July 6, 1963	23	783	669§	114	17.0	0	0.0
Illinois, State of							
July 1-July 31, 1966	24	9,617	8,469§	1,148	13.6	80	7.0
July 1-July 31, 1936	24	9,423	6,727§	2,696	40.1	1,193	44.3
Kansas City, Missouri							
July 1-July 31, 1980	25	598	362§	236	65.2	157	66.5
Memphis, Tennessee							
July 1-July 31, 1980	26	817	711§	106	14.9	83	78.3
New York, New York							
July 22-July 28, 1972	27	2,319	1,592§	727	45.7	10	1.4
Aug 31-Sept 7, 1973	27	2,242	1,808¶	434	24.0	22	5.1
June 30-July 6, 1872	27	1,569	769**	800	104.0	212	26.5
July 24-July 30, 1892	27	1,434	1,081**	353	32.7	231	65.4
Aug 9-Aug 15, 1896	27	1,810	809**	1,001	123.7	671	67.0
St. Louis, Missouri							
July 1-July 31, 1980	25	850	542§	308	56.8	122	39.6
July 9-July 14, 1966	28	543	240††	303	126.2	182	60.1

* Deaths during previous 2 weeks.
† Deaths during same period the following year.
§ Deaths during same period the previous year.
¶ Eight times the daily average of September 1973 deaths.
** Deaths during previous week.
†† Based on deaths during previous 8 days.

increase in thrombotic and embolic stroke in hot weather (32,33). Moreover, the consistency of the finding of excess stroke mortality during heat waves in different years and in different locations argues that the association between hot weather and stroke is a real one.

The frequency of deaths attributed to heart disease also increases during heat waves (Table 2), mainly due to an increase in deaths attributed to ischemic heart disease. The cause-specific death rate has increased in different heat waves by amounts ranging from about 7% to 55%, accounting for about 10%-40% of heat-wave-associated deaths (23,24,26,28-30).

As is true of the increase in deaths from stroke during heat waves, the great variability in the proportion of heat-wave-associated deaths designated as cardiac suggests that there are a substantial number of errors in death-certificate diagnoses in this category. Nevertheless, the evidence mentioned above regarding increased coagulability of blood in heat-stressed persons lends plausibility to the idea that hot weather causes an increase in deaths from ischemic heart disease (32,33). Moreover, the increase in cardiac deaths also occurs consistently during heat waves. Thus, it appears unlikely that the association between heat and death from ischemic heart disease is completely spurious.

It is possible that some of the heat-wave-associated deaths attributed to stroke or ischemic heart disease are actually misclassified deaths from heatstroke. This situation could arise because of problems in postmortem diagnosis. The recognition of heatstroke affecting a living patient who has characteristic neurologic findings and a very high body temperature presents few difficulties for the average clinician, especially if anhidrosis (greatly reduced or absent sweating) is present. However, heatstroke can progress rapidly to death, often within a few hours of the onset of symptoms. Duration of illness was less than 24 hours for 70% of patients in one study of 90 fatal heatstrokes (34). Thus, many persons who develop the disease die before they can be found and brought to medical attention. In the United States such relatively sudden out-of-hospital deaths are usually referred to the local coroner or medical examiner for a determination of cause of death, but no detailed postmortem examination of the body may be done. Under such circumstances the possibility exists that some heatstroke victims examined because of relatively sudden, unattended death are diagnosed as having died from other, more common causes (e.g., stroke, myocardial infarction) that can appear to be similar (35).

Postmortem temperature measurement can be useful in the diagnosis of heatstroke. During hot weather in some jurisdictions, the temperature of each body referred to the medical examiner is routinely measured, either by an investigator in the field or by the morgue attendant. A

TABLE 2. Percentage increase in selected causes of death and percentage of excess deaths during heat waves attributable to these causes, selected heat waves, United States, 1934-1983

DISEASE CATEGORY Heat wave location/period	Reference	Disease (author's words)	Percentage increase over control period	Percentage of heat wave deaths attributable to this cause
CEREBROVASCULAR				
Kansas State/July 1-31, 1934	30	Cerebral hemorrhage and softening	54.2	11.1
Illinois State/July 1-31, 1936	30	Cerebral hemorrhage and softening	39.2	6.0
Detroit, MI/May 12-18, 1962	23	Cerebrovascular accident	103.8	26.0
Detroit, MI/June 23-July 6, 1963	23	Cerebrovascular accident	81.9	51.8
Illinois State/July 1-31, 1966	24	Vascular lesions of central nervous system	26.3	20.1
New York, NY/July 2-15, 1966	29	Cerebrovascular accident	27.2	6.4
St. Louis, MO/July 9-14, 1966	28	Cerebral accident	53.3	7.0
CARDIAC				
Kansas State/July 1-31, 1934	30	Diseases of the heart	22.5	12.5
Illinois State/July 1-31, 1936	30	Diseases of the heart	40.8	25.2
Detroit, MI/May 12-18, 1962	23	Heart disease	14.0	18.3
Detroit, MI/June 23-July 6, 1963	23	Heart disease	6.9	15.8
Illinois State/July 1-31, 1966	24	Arteriosclerotic heart disease	13.3	36.1
New York, NY/July 2-15, 1966	29	Arteriosclerotic heart disease	40.8	41.5
St. Louis, MO/July 9-14, 1966	28	Cardiovascular disease	55.4	20.0
Memphis, TN/July 1-31, 1980	26	Cardiovascular*	40.0	84.9
Latium, Italy/July 1-31, 1983	39	Cardiovascular disease†	58.7	90.4
RESPIRATORY				
Kansas State/July 1-31, 1934	30	Pneumonia, all forms	74.6	2.5
Illinois State/July 1-31, 1936	30	Pneumonia, all forms	21.9	2.0
Detroit, MI/May 12-18, 1962	23	Respiratory	0.0	0.0
Detroit, MI/June 23-July 6, 1963	23	Respiratory	42.9	5.3
New York, NY/July 2-15, 1966	29	Respiratory	84.2	13.5
St. Louis, MO/July 9-14, 1966	28	Pulmonary disorders	27.8	3.3

* May include cerebrovascular deaths.
† Includes cerebrovascular deaths.

postmortem temperature of ≥ 106 F measured soon after death is a useful indicator of heatstroke, because core temperature changes relatively little during the first 1-3 hours after death, especially if the ambient temperature is not particularly low. The possibility of false-positive and false-negative results must be considered, however, since the core temperature of a cadaver eventually approaches that of its surroundings. In time, the body of a person who died of heatstroke will cool if the ambient temperature is lower than that of the body core and can rise if ambient temperature exceeds core temperature (36-38).

Stroke and other types of cardiovascular disease taken together may account for as much as 90% of the excess mortality noted during heat waves (26,39). Nevertheless, numbers of deaths from other causes have also been reported to rise. A clearly defined period of excess death due to respiratory causes corresponding to the July 1966 heat wave in the United States is apparent from national mortality statistics (40). In New York City, respiratory deaths rose 84% and accounted for 14% of the excess mortality attributed to the heat wave (29). However, in other heat waves respiratory deaths have not contributed importantly to excess mortality, generally accounting for 5% or less of such deaths (23,30). Currently, there is no clear pathophysiologic explanation for how an increase in respiratory deaths could occur from a heat wave.

There was a striking increase of 139% in the number of homicides committed during a 2-week period of hot weather in New York City in 1966. However, increases of similar magnitude have not been demonstrated subsequently, and, in any case, increased numbers of homicides accounted for less than 2% of the mortality excess during this heat wave (29).

Many heat-wave-associated deaths are not a clear and direct result of an overwhelming heat stress (heatstroke), nor do they fall into any of the other categories of disease mentioned above. They are seen in the form of apparently excess deaths from a broad variety of underlying causes (e.g., nephritis, diabetes) that do not have any obvious relationship to the heat. Mortality excesses in each of these categories do not occur consistently in every heat wave. Moreover, each specific diagnosis tends to account for a relatively small proportion of the excess death (23,29,30). It has been suggested that heat-wave-related mortality in this broad group of categories may reflect an ability of heat stress to precipitate death for debilitated persons who are ill from a wide variety of chronic diseases and would die in the near future anyway. As evidence of this, Lyster presented weekly totals of deaths occurring in Greater London and the rest of England's South-East Region before, during, and after the summer heat waves in 1975 and 1976. Mortality increased during both periods of severe heat in both geographic areas, but the

increases were followed by several weeks of seemingly lower-than-normal mortality (41). In heat waves before and since, however, such a phenomenon has been sought, but not observed. Henschel presented data showing that the average daily death rate in St. Louis was about the same before and after the 1966 heat wave, and Ellis et al. reported the absence of a deficit of deaths following a heat wave in New York in 1972 (27,28). Similarly, there was no substantial fall in mortality following the excess deaths resulting from the 1980 heat wave in St. Louis and Kansas City, Missouri (25). Thus, a depression in the crude mortality rate following a heat-wave-induced elevation is by no means a universal phenomenon.

Heat-Wave-Associated Morbidity

Nonfatal illness resulting from heat waves has been less well quantified than has heat-wave-related mortality. During the July 1980 heat wave, hospitals in St. Louis and Kansas City, Missouri, admitted 229 and 276 patients, respectively, with nonfatal illnesses thought by the attending physician to be related to the heat (25). In Memphis during the same period, there were 483 visits to emergency rooms for heat-related illness. Loss of consciousness was a frequent complaint, affecting almost half of the patients seen at City Hospital in Memphis. Dizziness, nausea, and cramps were other common symptoms. The proportions of the illnesses diagnosed for the 471 patients for whom diagnosis was known were as follows: heatstroke, 17%; heat exhaustion, 58%; heat syncope, 4%; heat cramps, 6%; and other heat-related illness, 15% (26).

Indirect measures of morbidity also rise with the heat. In July 1980 in St. Louis and Kansas City, Missouri, emergency-room visits rose 14% and 8%, respectively. The respective increases in overall hospital admissions were 5% and 2% (25).

Determinants of Risk

Variation in Heat-Related Health Effects over Time

The public health impact of heat at any given time depends not only on the weather conditions at that time, but also on previously existing conditions. A good example is the delay observed between the onset of the heat wave and the appearance of substantial adverse effects on public health. Unusually high temperatures on several days in succession are required to produce a noticeable increase in mortality, and heat waves lasting less than 1 week result in relatively few deaths. The importance of sustained hot conditions is also illustrated by the observation that heat waves in which relatively little nighttime cooling occurs (i.e., those in which daily minimum temperatures are especially elevated) are particularly lethal (27,41,42).

Over greater periods of time, however, hot weather seems to lose some of its virulence. Acclimatization of individuals to heat stress is a phenomenon that has been well documented by means of physiologic experimentation (43,44). Populations, too, seem to acclimatize to the heat over the course of a summer (45). Thus, heat waves in the northern hemisphere occurring in August and September seem to be less lethal than those occurring in June and July (46). During a sustained heat wave, after an initial dramatic increase, the number of deaths tends to return toward baseline, even though the temperature may remain elevated (45). This fall in crude mortality may result not only from acclimatization, but also from earlier deaths of susceptible persons, decreasing their number in the population at risk (23).

Urbanization and Risk

Heat waves cause a disproportionately severe health impact in cities, to a large extent sparing more rural and suburban areas. In July 1980, deaths in St. Louis and Kansas City, Missouri, were 57% and 65% higher, respectively, than in July 1979. In contrast, there was an excess mortality of only 10% in the remainder of Missouri, which is largely suburban and rural (25). This trend is not a recent development. In a review of deaths caused by heat and registered in the United States from 1900 to 1928, Shattuck and Hilferty found that the rate of heat-related deaths was substantially higher in urban than in rural areas (47). In a later work, the same investigators found the effect of heat on death rates increased markedly with increase in the size of a city, suggesting a sort of "dose-response" effect of urbanization (48).

One reason health effects of hot weather may be more extensive in cities is that temperatures there may actually be somewhat higher than in surrounding rural and suburban areas. During the 1980 heat wave, the daily maximum temperature averaged 2.5 C higher and the daily minimum temperature averaged 4.1 higher at the Kansas City downtown airport than at the suburban Kansas City International Airport (25).

The concept of the urban "heat island" has also been invoked to explain the disproportionate severity of the health impact of heat in cities. The masses of stone, brick, concrete, asphalt, and cement that are typical of urban architecture absorb radiant heat energy from the sun during the day and radiate that heat during nights that would otherwise be cooler. In many cities there are relatively few trees to provide shade. Tall city buildings may effectively decrease wind velocity, thereby decreasing the contribution of moving air to evaporative and convective cooling (2).

The relative poverty of some urban areas is another factor that may contribute to the severity of urban heat-related health effects (25). Poor people are less able to afford cooling devices and the energy needed to run them.

One report from Italy suggests that an urban predominance of heat-related health effects may not be universal. During a heat wave in July 1983 in Latium Region, one of 20 regions into which Italy is divided, mortality recorded at various inpatient facilities (hospitals and clinics) increased 49% over the previous year in the area outside of Rome, but only 25% in Rome itself (39). The reasons for this anomalous finding are not yet clear.

There is considerable variation among different cities with regard to susceptibility to hot-weather-related health effects. For example, summer temperatures that would not be considered unseasonably high in Phoenix, Arizona, have occurred in St. Louis, Missouri, and caused a severe, adverse impact on public health. In July 1980 in St. Louis, 122 deaths and 229 hospitalizations and an increase in total mortality of

57% over the previous year were attributed to the heat (25). During that period, however, the average daily maximum temperature in St. Louis was 95.4 F, 12.2 F lower than the normal July daily maximum temperature in Phoenix; the average daily minimum temperature in St. Louis was 74.5 F, 3 F lower than Phoenix's normal daily minimum temperature. The highest temperature recorded in St. Louis during the heat wave was 107 F, only 2.2 F higher than the expected (normal) maximum temperature of 104 F on any given July day in Phoenix (49). Even after taking the higher humidity of St. Louis into account, its July 1980 temperatures were approximately those of an average July in Phoenix (1). It is noteworthy that neither excess mortality nor prominent heat-related health effects were noted in Phoenix in July 1980 despite temperatures that averaged 4.4 F above normal for that city and a monthly high temperature of 115 F (20).

The reasons for the differences in heat sensitivity of various cities have not been studied extensively. Possible explanations include differences in population age structure and acclimatization, architectural style, building materials, and use of air conditioning.

High-Risk Groups

The overall mortality increase observed during the heat wave disproportionately affects the elderly. During a heat wave in July 1983, deaths in Rome, Italy, increased 23% overall, but increased 35% among persons > 64 years of age (39). The increase in mortality in Greater London resulting from a heat wave in 1975 occurred almost exclusively among persons ≥ 65 years of age (50). In New York City in the summer of 1966, deaths among persons ages 45-64 increased substantially, but the increase in deaths of persons > 80 years of age was far greater. In this study, the investigators also judged that excess deaths among persons ≥ 80 years of age began earlier in the heat wave than those among persons ages 45-64 years, possibly indicating greater heat sensitivity of the older group (29).

Deaths specifically designated by physicians as having been caused by the heat also occur with a disproportionately high frequency among the elderly. This trend is easily seen in Figure 2, a graph of age-specific rates for heat-related mortality in the United States in the period 1968-1980. The rate of heat-related mortality is lowest in late childhood. It then increases monotonically throughout the teenage and adult years, with the slope of the curve increasing particularly rapidly as old age is approached. This pattern is not a new one, since Shattuck and Hilferty observed essentially identical trends associated with heat-related deaths in Massachusetts in 1900-1930, in New York in 1900-1928, and in Pennsylvania in 1906-1928 (47).

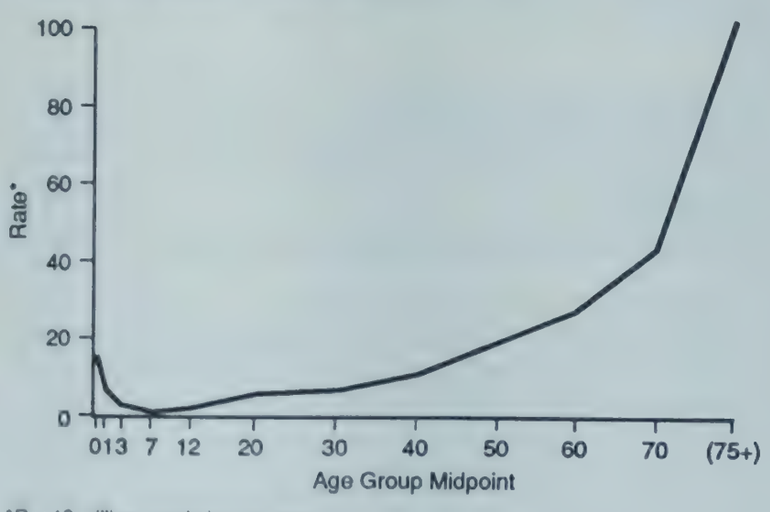
The elderly are also at increased risk of acquiring illness that meets strict diagnostic criteria for heatstroke. A 1980 study of persons with nonfatal heatstroke in St. Louis and Kansas City revealed that 71% were > 65 years of age, although this age group made up only about 15% of the population at risk (25). Other studies have consistently confirmed the susceptibility of the elderly (26,28,46).

The predisposition of the elderly to health effects of the heat may partially reflect impaired physiologic responses to heat stress. Vasodilation in response to the heat requires increased cardiac output, but persons > 65 years of age are less likely to have the capacity to increase cardiac output and decrease systemic vascular resistance during hot weather (51). Moreover, the body temperature at which sweating begins increases with increasing age (52). The elderly are more likely than younger persons to have chronic disease or to be taking medications (e.g., major tranquilizers and anticholinergics) that can increase the risk of heatstroke (53,54).

Finally, old people perceive differences in temperature less well than do younger persons (55). They may, therefore, less effectively regulate their thermal environments.

At the other extreme of age, the rate of physician-diagnosed heat-related death is higher for babies and young children <5 years of age than for older children, as shown in Figure 2. The magnitude of this increased risk, however, is nowhere near as great as for elderly persons. There was no detectable increase in mortality for the age group 0-4 years in Greater London during the June-July 1975 heat wave (50). Only one of the 83 persons who died of heat-related causes in Memphis, Tennessee, in July 1980 was < 20 years of age (a baby in the first year of life) (26). No cases of fatal or nonfatal heatstroke were found to have occurred among persons aged 0-18 years in St. Louis or Kansas City during the July 1980 heat wave, despite careful case-finding efforts in pediatric hospitals and medical examiners' offices (25). Nevertheless, Henschel and others found that four of 182 persons who died of heat-related illness were babies < 1 year of age during the July 1966 heat wave in St. Louis (28). The small but definitely increased risk of death from heat for babies and young children is more clearly seen in summaries of state and national data compiled over a number of years than in studies of specific heat waves in individual cities.

FIGURE 2. Rates of death attributable to heat (ICD E900), by age, United States, 1968-1980



*Per 10 million population per year.

Sources: National Center for Health Statistics and the Bureau of the Census.

Other observations document the sensitivity to heat of the very young. Healthy babies kept in hot areas have been found to run temperatures as high as 103 F, and mild fever-causing illnesses of babies may be tipped over into frank heatstroke by heat stress. Children with congenital abnormalities of the central nervous system and with diarrheal illness appear to be particularly vulnerable (56,57). Parents may contribute to risk by failing to give enough hypotonic fluid during the heat and by dressing the child too warmly (57,58).

U.S. national figures show that males have an increased risk of heat-related death compared with females in the teenage years and during early and middle adult life (Table 3). This difference may reflect a tendency toward greater heat exposure and exertional heat stress among males from occupational and leisure activities, but definitive epidemiologic data on this point are lacking. At the extremes of age, there is little difference between the sexes in rates of heat-related deaths.

TABLE 3. Death rates,* by sex and age and rate ratios,† attributable to heat, United States, 1968-1980

Heat (*International Classification of Diseases* E900)

Age	Male	Female	Rate ratio
<1	14.8	15.5	1.0
1	8.9	5.5	1.6
2-4	8.9	2.0	1.6
5-9	0.6	0.3	1.9
10-14	1.3	0.4	3.3
15-24	9.3	1.1	8.2
25-34	10.5	1.8	6.0
35-44	16.9	3.6	4.6
45-54	29.2	7.8	3.8
55-64	38.2	15.2	2.5
65-74	52.5	33.4	1.6
>75	117.8	92.7	1.3

* Per 10 million population/year.
† Rate ratio = male/female.

Interestingly, many studies of heat waves have demonstrated greater numbers of heat casualties among women than among men. During the 1966 heat wave in New York City, deaths among women were 50% greater than expected, but deaths of men were increased by only 25% (29). In July of the same year in St. Louis, 59% of heat-related deaths occurred among women (28). In July 1980 in Memphis, 61% of 83 persons who were diagnosed by a physician as having died from the heat were women (26). In Latium Region, Italy, women accounted for 65% of fatal cases meeting specific diagnostic criteria for heatstroke (39).

The probable reason for an apparent excess of women among heat fatalities despite the generally higher age-specific rates of heat-related mortality among men is that age confounds the association of female sex with death due to heat. Elderly populations are the ones at greatest risk, and there are substantially more women than men among the elderly (59). The existence of such confounding was demonstrated in a heat-wave study in which age-adjusted rates of heatstroke were virtually identical for the two sexes despite a predominance of female study subjects (25).

Heat-related health effects are disproportionately severe in areas of low socioeconomic status. In 1966 in St. Louis, the death rate rose most dramatically in areas of low median family income in which there was substantial crowding (high number of persons/room) (29). In 1980 in St. Louis and Kansas City, Missouri, the heatstroke rate in census tracts in the highest socioeconomic quartile was about one-sixth that in tracts in the lowest quartile. The rates were intermediate in the tracts of intermediate socioeconomic status (25). Factors leading to the relatively low incidence of heat-related health effects in well-to-do areas may include availability of air conditioning, abundance of trees and shrubs that provide shading, and access to health care.

In several studies the rates of heat-related illness have been higher for blacks than for whites. In 1980 in Texas, the heat-related death rate was 21.1/million for blacks and 8.1/million for whites (46). Age-adjusted heatstroke rates were three to six times higher for minority races (principally blacks) in St. Louis and Kansas City in July 1980 (25). The association of black race and relatively low socioeconomic status may well account for the disproportionately high heatstroke rate for blacks in the United States. No biologically based vulnerability of any particular race has been shown.

Persons with a history of prior heatstroke have been shown to maintain thermal homeostasis in a hot environment less well than otherwise comparable volunteers (60). Whether a heatstroke damages the brain's thermoregulatory apparatus or thermoregulative abnormalities antedate the first heatstroke is not known. However, persons with a history of heatstroke should be considered at risk of a recurrence.

Obesity is an important factor affecting heat tolerance. Obese subjects exercising in a hot environment show a greater increase in rectal temperature and heart rate than do lean subjects (61-62). The insulating effect of subcutaneous fat impedes the transfer of metabolic heat from core to surface. Soldiers in the U.S. Army who died of heatstroke during basic training during World War II were much more obese than their peers (63). However, obesity may not importantly influence the rate of heatstroke for the elderly, largely sedentary population that is at greatest risk during a heat wave (53).

Persons with other, less common conditions may also tolerate the heat poorly. These conditions include congenital absence of sweat glands and scleroderma with diffuse cutaneous involvement. In both conditions, perspiration is markedly diminished, resulting in impaired thermoregulation in a hot environment (64,65).

Some drugs predispose to heatstroke. Neuroleptic drugs (e.g., phenothiazines, butyrophenones, and thioxanthenes) have been particularly strongly implicated. Phenothiazine-treated animals survive in a hot environment for shorter periods than controls, and heatstroke occurs with increased frequency among patients taking these drugs (53,66). Neuroleptics appear to sensitize both to cold and heat (66).

Anticholinergic drugs decrease heat tolerance in laboratory tests of human volunteers. Persons treated with anticholinergics while exposed to heat have been reported to have a decrease or cessation of sweating and a rise in rectal temperature (67). Many commonly used prescription drugs (e.g., tricyclic antidepressants, some antiparkinsonian agents) and nonprescription drugs (e.g., antihistamines, sleeping pills) have prominent anticholinergic effects, and in one study the use of such drugs was more common among heatstroke victims than among controls (53). The likely mechanism of action appears to be inhibition of the ability to perspire.

Certain stimulant and antidepressant drugs taken in combination or in overdose may induce the syndrome of heatstroke. Severe hyperthermia has been reported to result from an amphetamine taken in overdose, an amphetamine

taken with a monoamine oxidase inhibitor, and a tricyclic taken in combination with a monoamine oxidase inhibitor (68-70).

Methodologic Problems of Epidemiologic Studies

The literature on heat-wave-related morbidity and mortality has been complicated by the fact that different researchers have studied different health outcomes. In some heat waves that cause substantial excess mortality, relatively few or none of these deaths are certified as having been caused by heat. Since physician-designated heat-related deaths are often so few in comparison to the magnitude of the total increase in mortality, some investigators have chosen to study the total increase in mortality itself as the health outcome of importance. In such studies the number of deaths has been studied in relation to the results of meteorologic measurements made at a local weather station. Since the administrative mechanisms for recording the mere occurrence of a death on a given date are fairly dependable in developed countries, the measure of the health outcome being studied (death on a particular day) is almost exact. Nevertheless, the weather station from which the data are taken may be at a site, such as an airport, miles away from the area in which most deaths occur. Even if readings are taken within the area inhabited by the population at risk, they are outdoor measurements that do not necessarily reflect the variable conditions within dwellings and other buildings in which most of the deaths occur. Since such studies also fail to take into account other host and environmental risk factors, only very limited conclusions can be drawn from their findings.

Many authors have studied heat-wave-associated mortality using information provided on death certificates, comparing deaths occurring during a heat wave with those during a control period. Apparent excess death attributed to a variety of diagnostic entities (e.g., stroke, ischemic heart disease) has been studied, not just that corresponding to clear-cut heat-related illness (e.g., heatstroke). These studies have yielded interesting findings, but the well-known imprecision in certain of the data listed on death certificates leads to corresponding imprecision in study results. In particular, physicians' criteria for diagnosing various causes of death vary over time and in different locations.

In an attempt to deal specifically with morbidity and mortality that are clearly due to the heat—excluding cases of illness and death that could have occurred even in the absence of heat—some investigators have limited their studies of disease to cases classified as "heat-related." This term generally refers to a physician's determination that an illness or death was in some way related to environmental heat, and that is the sense in which the term is used in this chapter. But even defined in this way, the term is somewhat ambiguous. Heat can produce several distinct syndromes, all of which are 'heat-related." Moreover, heat-related death in some studies refers only to deaths in which environmental heat is judged to be the underlying cause of death, but in other reports deaths for which heat was only a contributing factor are also included. Moreover, the use of this categorization in diagnosis and coding of the cause of death may vary greatly from region to region. Writing about the 1966 heat wave, which caused severe health consequences in New York City and St. Louis, Schuman observed that 130 deaths in St. Louis were attributed to "excessive heat and insulation" but that in New York City "only a handful of deaths were so coded, preference being assigned . . . to underlying circulatory and degenerative conditions" (29).

In an effort to limit parts of their investigation to the study of a clear-cut illness caused by heat, researchers investigating the effects of the 1980 heat wave in St. Louis and Kansas City, Missouri, defined the following persons as having heatstroke:

> Patients with a presenting temperature (measured anywhere on the body) greater than or equal to 41.1 C (106 F); patients with documented temperature greater than or equal to 40.6 C (105 F) if altered mental status or anhidrosis was also present; and those pronounced dead on arrival at the hospital or medical examiner's office if the body temperature . . . was greater than or equal to 41.1 C (106 F) (53).

Other studies undertaken since that time have defined heatstroke similarly (39,71). Strict definitions could also be developed for the study of other outcomes whose direct cause is the heat (i.e., heat exhaustion, heat syncope, heat cramps). Such definitions do not necessarily help the clinician attempting to diagnose the case of a particular patient. Their usefulness lies in their value as entry criteria for epidemiologic studies of groups of patients, enabling the investigator to explain precisely which clinical entities have been studied when "heat-related" illness is the subject of the study. In this manner, future investigators will be better able to clarify and quantify the health consequences of heat.

Prevention of Adverse Health Effects Caused by Heat

Timing of Preventive Measures

In most parts of the United States, heat waves severe enough to threaten health do not occur every year, and several relatively mild summers may intervene between major heat waves. The erratic occurrence of heat waves hinders effective planning of prevention efforts. It may be administratively difficult for health departments to plan for adequate resources to be available if needed, but not wasted if no heat wave occurs.

Although long-term weather forecasts (i.e., those done some months in advance of the event) cannot reliably predict periods of severe heat, near-term forecasts of hot weather several days in advance are increasingly accurate. Could one also forecast the extent of mortality and morbidity expected to result from anticipated hot weather? Even 1 or 2 days of advance warning regarding the probable extent of heat-related adverse health effects would be of use in planning for their prevention.

"Apparent Temperature," also known as "heat index" (one of the indices of human heat stress discussed above), has been proposed as a guide for how hazardous to health the anticipated weather may be. However, the index was not developed for this specific use. The hazard posed by heat stress depends not only on its magnitude at a given moment, but also on how it has varied over time. Moreover, this index in no way takes into account the great variation in

heat sensitivity of different regions. Thus, Apparent Temperature, independent of geographical location and antecedent meteorological events, will probably not be found to be a very useful predictor of the extent of heat-related health effects to be expected in a population at risk (1,2,6).

Several authors have attempted to develop mathematical models to quantify the increase in numbers of deaths to be expected for a given degree of temperature increase. These formulae have taken into account such factors as the usual seasonal trends in mortality, acclimatization, and the age structure and previous hot weather exposure of the population at risk. Currently available mathematical models have been fitted retrospectively to past mortality and meteorologic data. They are reasonably in accord with the observations from which they were developed. However, none of these models has yet demonstrated its usefulness in the prospective prediction of heat-related adverse health effects (42,45,72). This is an important area of current research.

In the absence of reliable prediction, early detection of important adverse health consequences of heat could provide public health professionals with useful information, allowing them to mobilize resources for prevention relatively early in an epidemic of heat-related illness. A large increase in the case load of the local medical examiner that is unexplained by any other disaster has been proposed as an early indicator of severe heat-related health effects in a community. This proposal was based on 1980 data from two midwestern cities showing that the number of cases reported to medical examiners increased to a proportionately greater extent than did other indirect measures of the heat's impact on public health, including total mortality, emergency-room visits, and hospital admissions. Moreover, the total number of medical-examiner cases is much more easily and rapidly available than these other statistics. Even the time required for postmortem diagnosis does not delay data collection (35). Although prospective evaluation has not yet established the degree of utility of this sort of surveillance, this should not discourage state and local health departments from further evaluation of the method within their jurisdictions. There are as yet no firm criteria regarding just how much of an increase in case load should trigger implementation of prevention programs.

Content of Prevention Programs

Programs to prevent heat-related illness should concentrate on measures whose efficacy is supported by empirical data. Many heat-illness prevention efforts have centered around the distribution of electric fans to persons at risk. However, study of the 1980 heat wave in Missouri did not show a significant protective effect of fans (53). This finding is consistent with theoretical predictions and empirical data showing that as air temperature rises toward about 99 F—the exact value depends on the humidity and other factors—increased air movement ceases to lessen heat stress. At even higher temperatures, increased movement of air may actually exacerbate heat stress (1-3,6). Although further epidemiologic studies are required to properly evaluate the preventive efficacy of fans, they should probably not be used in situations in which established indices of heat stress suggest they might be harmful.

Air conditioning effectively prevents heatstroke and may decrease the incidence of other adverse health effects of heat waves. In one study, the presence of 24-hour air conditioning in the home reduced heatstroke risk by 98%. In addition, just spending more time in air-conditioned places (regardless of whether there was a home air conditioner) was associated with a fourfold reduction in heatstroke (53). These findings suggest that air-conditioned shelters are an effective means of preventing heatstroke. Persons at high risk who do not have home air conditioners may benefit from spending a few hours each day in an air-conditioned environment. The maintenance of adequate hydration is important in preventing heat-related illness. Increases in the body temperature of heat-stressed volunteers were lessened when fluid losses were frequently replaced (73). Moreover, taking extra liquids has been associated with decreased risk of heatstroke (53). More fluid than the amount dictated by thirst may be required to fully offset the increased fluid losses that occur during hot weather (73,74). Thus, unless there is a medical contraindication, persons at risk from the heat should be advised to make a special effort to increase the amount of liquid they consume.

Adequate intake of salt with meals is important. Although salt supplementation with tablets may be important in preventing electrolyte imbalances for carefully selected individuals who must tolerate intense heat for prolonged periods (18), it is of doubtful benefit in preventing heat-related illness in the general population (53). Furthermore, such supplementation may be harmful for persons with certain chronic illnesses in which a high sodium intake is undesirable (e.g., persons with hypertension, congestive heart failure). Therefore, salt tablets should not be recommended for consumption by the general population during a heat wave.

Persons at high risk should be advised to reduce activity in the heat, since such behavior appears to have protected against heatstroke in one study (53). Conversely, athletic exertion in the heat substantially increases risk, although risk does not increase as much for persons who have become acclimatized by training in the heat (63).

Target Groups

To be maximally effective, programs for the prevention of heat-related illness should be directed toward groups known to be at particularly high risk. Cities—especially low socioeconomic-status, inner-city areas—are particularly appropriate targets for prevention efforts. The elderly should receive special attention, since old age is one of the factors most strongly associated with increased risk of heatstroke or death from other causes during the heat. As much as possible, special living facilities for the elderly and institutions such as nursing homes and hospitals in which many elderly persons are to be found should be air conditioned during severely hot weather. The elderly living at home should not be forgotten, however, since they may be at even greater risk than those in institutions (75). Parents should be made aware of the increased heat sensitivity of babies and children < 5 years of age. Patients taking neuroleptic or anticholinergic drugs should be counseled regarding their possible increased sensitivity to heat.

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Cold Environments

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Background and Nature of Physical and Physiologic Aspects of Cold Exposure

Scope of the Problem

Unlike heat waves, periods of sustained, unusually cold winter weather do not characteristically cause a clear-cut rise in numbers of daily deaths from all causes. Nevertheless, the cold causes mortality and severe morbidity of considerable public health importance. In fact, if one considers only U.S. deaths certified by physicians as being temperature-related, the average number of deaths attributed to the cold (about 700 yearly) is substantially higher than the average yearly number attributed to the heat (about 200) (1).

Physical and Meteorologic Factors

Air temperature, humidity, wind speed, and radiant heat energy are the four environmental factors of greatest importance in determining human heat stress; they also determine cold stress. However, these factors differ in relative importance in causing stress from heat and cold. Air movement is far more effective in facilitating convective heat loss from the body in cold conditions than in warm ones. On the other hand, since the regulation of perspiration is not an important physiologic mechanism for maintaining body temperature in the cold, changes in humidity do not affect cold stress as much as they do heat stress. Radiant heat emitted by indoor heating devices (e.g., stoves, radiators) may substantially ameliorate indoor cold stress. The sun, however, transmits radiant heat to the earth's surface less efficiently in winter than in summer, resulting in a relative decrease in the importance of outdoor variations in radiant heat in the determination of cold stress. Thus, for most purposes, air temperature and wind speed are the two factors most important in determining thermal stress under cold conditions, particularly outdoors (2,3).

A widely used "wind-chill" index formulated by Siple and Passel in 1945 relies only on air temperature and wind speed in predicting the cold stress resulting from specific meteorologic conditions (3). Although widely applied and generally useful, this index has inherent inaccuracies at the extremes of wind speeds, and alternative schemes have been proposed (4,5). A relatively standard wind-chill equivalent temperature guide is shown in Table 1.

Adaptive Mechanisms

The principal immediate adaptive physiologic responses to the cold are shivering and vasoconstriction. Muscular activity related to shivering causes increased metabolic heat production. Peripheral vasoconstriction causes a rerouting of some blood away from cutaneous and other superficial vascular beds toward deeper tissues, where the blood's heat can be more readily retained. In addition, blood is rerouted from the superficial veins of the limbs to the venae comitantes of the major arteries. There, through a 'countercurrent" mechanism, arterial blood warms venous blood before its return to the core and cools arterial blood, so that it gives up less heat when it reaches the periphery. The result is a fall in the temperature of superficial body parts in defense of core temperature. The difference between skin and core temperatures is thus an approximate measure of the efficacy of vasoconstriction (6,7).

Cold Weather and Mortality

In the United States, mortality usually peaks in midwinter and reaches a low point in late summer (Figure 1). The amplitude of the cyclic rise and fall in mortality is quite large, resulting in the occurrence of tens of thousands more deaths in January than in September. Thus, the increased numbers of deaths in the winter substantially exceed the number of deaths certified each year as having resulted from the cold (1). A similar seasonal rise and fall in death rate occurs in other countries in the temperate regions of both the northern and southern hemispheres. Of course, the seasonal patterns of these two hemispheres are 6 months out of phase, the death rate being maximal in winter in each hemisphere (6). Nevertheless, winter "cold snaps" (several days or more of unusually cold weather) do not seem to cause the sudden, striking increases in overall mortality that summer heat waves cause. Therefore, investigators have had to use statistical analysis of daily temperature and mortality data to

TABLE 1. Wind-chill equivalent temperatures for a reference wind speed of 4 miles per hour (1.79 meters per second)

Temperature (Degrees F)	Actual wind speed (in miles per hour)						
	4	5	10	20	30	40	50
40	40	37	28	18	13	10	9
35	35	32	22	11	5	2	1
30	30	27	16	4	−2	−6	−7
25	25	22	10	−3	−10	−14	−15
20	20	16	4	−10	−18	−22	−23
15	15	11	−3	−18	−25	−29	−31
10	10	6	−9	−25	−33	−37	−39
5	5	1	−15	−32	−41	−45	−47
0	0	−5	−21	−39	−48	−53	−55
−5	−5	−10	−27	−46	−56	−61	−63
−10	−10	−15	−33	−53	−64	−69	−71
−15	−15	−20	−40	−60	−71	−77	−79
−20	−20	−26	−46	−67	−79	−85	−87

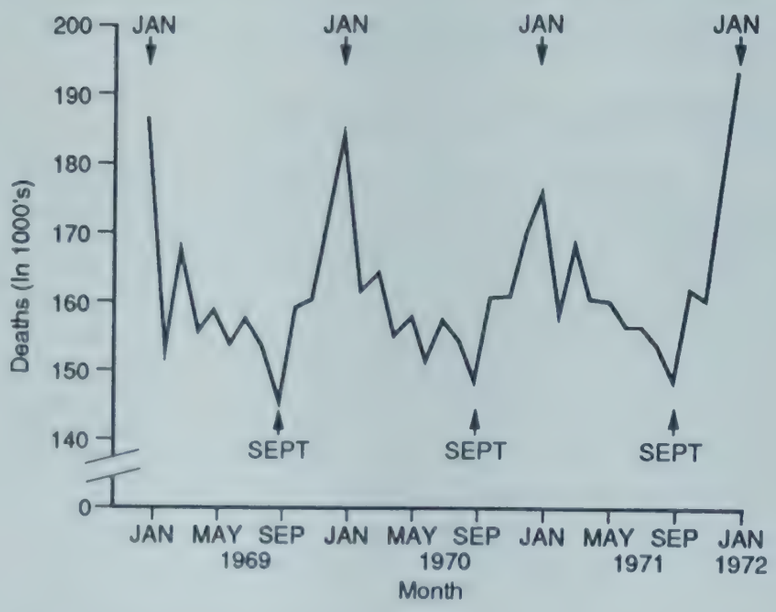
demonstrate the increase in numbers of deaths associated with the cold (8-11). This analysis is greatly complicated by the seasonal variation in mortality observed in countries of the temperate regions of both hemispheres.

The tendency for death to occur in the winter is most marked among the elderly and becomes increasingly prominent with increasing age. Moreover, for persons < 46 years of age the trend is reversed. For this group, death is more likely to occur in summer than in winter. Many of the major causes of death are associated with increases in mortality in the winter, among them diseases of the heart, cerebrovascular disease, pneumonia, influenza, and chronic obstructive pulmonary disease. In contrast, deaths from malignant neoplasms remain virtually constant throughout the year (12).

Because of the seasonal fluctuation in mortality, regression analyses based on daily observations show a significant negative correlation between mortality and temperature, that is, a direct association of high death rate with cold weather. The strength of this association does not necessarily imply that cold weather is the direct cause of all of the wintertime death increase. In fact several observations tend to indicate that this seasonal increase is not related to temperature. One is that the peaks and valleys in the U.S. mortality curve have not always appeared in January and September as they do now. In the early part of this century, the peak was usually in February or March and the nadir in June (13). Winter death increases occur even in states with relatively mild winters (e.g., Florida and Hawaii), and such increases are of approximately the same magnitude as those observed in states in which winter is characteristically harsh (e.g., Minnesota and Montana) (12). One epidemiologic analysis sought to control for the confounding effect of seasonal fluctations in mortality by limiting its observations to the months November through February. The death rate for men <65 years of age increased modestly on days with mean temperatures below the monthly mean, but there was little change in numbers of deaths among persons of both sexes ≥65 years of age, the group for which the seasonal mortality increase is most pronounced (12,14).

Nevertheless, the ability of the cold to cause severe illness and death should not be underestimated. Deaths from stroke, ischemic heart disease, and pneumonia may all increase as a direct result of the cold (10,11). Cold-related increases in blood pressure and coagulability may cause the reported increases in deaths from stroke and ischemic heart disease (15,16).

FIGURE 1. U.S. deaths, by month, January 1969-January 1972



Adapted from *Public Health and Preventive Medicine*, J. M. Last, ed., 12th Edition, 1986.

Hypothermia

The term "hypothermia" refers either to the unintentional or purposeful lowering of core body temperature. Hypothermia has been purposefully induced to decrease oxygen consumption during certain surgical procedures (6,7).

Unintentional hypothermia is usually the result of overexposure to the cold and is a problem of considerable public health importance. The latter is the only type of hypothermia discussed below.

Hypothermia is the only known cold-related illness with a substantial death-to-case ratio. It is thus reasonable to suppose that cases of hypothermia account for the great majority of deaths for which exposure to cold is certified as the underlying cause (E901, *International Classification of Diseases, 9th Revision)*. However, this supposition has not yet been verified.

Most authorities agree that hypothermia is clinically significant when core body temperature falls to ≤ 95 F (35 C). As body temperature drops, consciousness becomes clouded, and the patient appears confused or disoriented. Pallor results from intense vasoconstriction. Shivering occurs at first, but decreases markedly in intensity as body temperature falls further and hypothermia itself impairs thermoregulation. With severe hypothermia (body temperature < 86 F or 30 C), consciousness is lost, respiration may become imperceptibly shallow, and the pulse may not be palpable. At such low temperatures, the myocardium becomes irritable, and ventricular fibrillation is common. The patient may appear dead even though s/he may yet be revived with proper treatment (6). Persons found apparently dead in circumstances suggesting hypothermia should be treated for hypothermia until death can be confirmed (17). In particular, the potential for recovery of apparent victims of cold-water drowning should not be underestimated, since there have been reports of virtually complete recovery of patients who were without an effective heartbeat for periods as long as 2.5 hours (18).

Hypothermia can be primary or can result from thermoregulatory failure caused by other illness—particularly sepsis, myocardial infarction, central-nervous-system damage, or metabolic derangements. Secondary hypothermia has a worse prognosis than does primary hypothermia, probably because of the severe nature of the concomitant illnesses capable of producing hypothermia (19).

Controversy exists regarding the optimal method(s) for rewarming patients with hypothermia. Advocates of slow external rewarming contend that relatively rapid rewarming causes an abrupt release of vasoconstriction in acral body parts, resulting in a sudden influx of cold, acidotic blood into the core, which exacerbates the metabolic derangements of hypothermia. Further, the release of vasoconstriction is said to result in a relative hypovolemia that may precipitate shock (7,20). Advocates of rapid external rewarming counter that volume deficits and acidosis can be rapidly corrected by infusion of fluids and sodium bicarbonate, and that the best way to treat any further deleterious effects of the cold is rewarming itself (21,22).

The treatment of hypothermia under extreme circumstances (e.g., when intractable ventricular fibrillation occurs) is more standardized. In such circumstances rapid, invasive "core" rewarming by such methods as peritoneal or open mediastinal lavage or cardiopulmonary bypass is generally advocated (17,23,24).

No matter what method of rewarming is used, all but very mild hypothermia cases require intensive care, including respiratory support, electrolyte and acid-base disturbance correction, and intravascular volume optimization. Hypoglycemia should be checked for and corrected. In addition, the patient must be treated for any predisposing medical condition.

Local Tissue Injury Produced by Cold

Frostbite is damage to local tissue caused by that tissue's being frozen. This damage results from the mechanical effects of ice crystals, the denaturation of intracellular proteins caused by hypertonicity of the unfrozen tissue fluid, and subsequent microvascular occlusion in the affected area. Acral body parts—including hands, feet, ears, and nose—are most frequently affected. In mild cases, recovery is usually complete. However, in severe cases tissue viability is affected, gangrene develops, and amputation of affected tissues may be required. Optimal treatment includes rapid rewarming in a water bath with a temperature of 42 C and subsequent supportive care (2).

Prolonged exposure to cold conditions at above-freezing temperatures may also cause tissue injury. "Trench foot" and "immersion foot" result from prolonged (days to weeks) exposure to wet and cold conditions. Affected persons develop paresthesia, hypersensitivity to cold, and muscle weakness or atrophy. In severe cases there may be superficial gangrene. Pernio (chilblain) is superficial ulceration occurring on body parts (especially the legs) subjected to prolonged exposure to dry cold. Both dry- and wet-cold nonfreezing injuries are thought to result from a prolonged vasoconstrictive response to cold and consequent circulatory inadequacy to affected body parts (2).

Determinants of the Risk of Hypothermia

Situational Factors

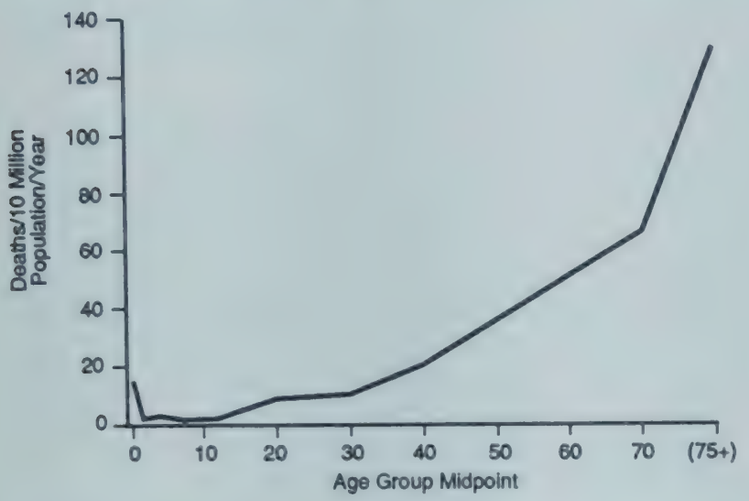
Unintentional hypothermia tends to arise in one of two sets of circumstances. One situation involves hypothermia affecting relatively young and generally healthy participants in outdoor sports that are often done in cold weather (e.g., hiking, camping, skiing). In this manner, an uncompromised host may be subjected to an overwhelming cold stress. Hypothermia may develop relatively rapidly, over a period of hours. Frostbite frequently accompanies hypothermia in this situation, since temperatures below freezing are generally involved. Factors that increase the likelihood of developing hypothermia include wearing insufficient clothing, getting clothing wet (which decreases its value as an insulator), and immersing oneself in cold water (in which the relatively high heat-conducting capability of water results in rapid loss of heat from the body). Hypothermia may impair the judgement of recreationists, causing them to remain in situations of dangerous cold stress or not to protect themselves adequately (6).

The second situation in which hypothermia commonly occurs involves a particularly vulnerable person who is subjected to only a moderate, indoor cold stress. A common example is that of an elderly person living in an inadequately heated home. In such circumstances hypothermia may not occur until days or weeks after the cold stress begins, and frostbite does not usually develop, since temperatures below freezing are not commonly involved (6). The risk factors in this situation are distinct from those involved in hypothermia among recreationists.

Hypothermia Involving the Elderly

The special vulnerability of elderly persons to hypothermia has been increasingly appreciated in recent years. After the first year of life, the rate of death due to effects of the cold increases with advancing age (Figure 2). In the United States, over half of the approximately 700 persons who die each year due to cold exposure are ≥ 60 years of age, although persons in this age group represent < 16% of the population (1,25).

FIGURE 2. Rates of death associated with cold (ICD E901), by age, United States, 1968-1980*



International Classification of Diseases

Adapted from *Public Health and Preventive Medicine*, J. M. Last, ed., 12th Edition, 1986.

The extent of morbidity from hypothermia among the elderly is less easily measured. A national wintertime survey of 1,020 persons ≥ 65 years of age conducted in Great Britain found that relatively few (0.58%) had hypothermic (≤ 35 C) morning deep-body temperatures, and none had hypothermic evening temperatures. However, a substantial number (9%) had near-hypothermic temperatures (≥ 35.5 C but < 35 C) (26). In contrast, 3.6% of 467 patients > 65 years of age admitted to London hospitals in late winter and early spring had hypothermia (27). That hypothermia is relatively common among elderly persons admitted to hospitals—although virtually absent in the community—has been interpreted as showing that most elderly Britons with hypothermia are quickly hospitalized.

The apparent sensitivity to cold on the part of the elderly may be due to physiologic factors. Collins and others found that a high proportion of persons ≥ 65 years of age failed to experience physiologically significant vasoconstriction in response to a controlled cold environment and that the proportion of such persons increased with the age of the cohort examined. Elderly subjects with abnormal vasoconstriction tended to have relatively low core temperatures (28). The basal metabolic rate declines substantially with age, requiring elderly people to battle cold stress from a relatively low level of basal thermogenesis (29), and the shivering mechanism of some older persons may be impaired (30). Voluntary muscular activity also releases heat, but the elderly are more prone than younger persons to debilitating chronic illnesses that limit mobility. Brown fat, a type of tissue whose principal purpose seems to be the generation of metabolic heat, is less abundant in old people than in children and younger adults (31).

Elderly persons appear to perceive cold less well than younger persons and may voluntarily set thermostats to relatively low temperatures (32). In addition, the rising cost of energy in recent years, together with the relative poverty of some elderly people, may discourage their setting thermostats high enough to maintain adequate warmth.

Drugs Predisposing to Hypothermia

Ethanol ingestion is an important predisposing factor for hypothermia. The great majority of patients in many case series on hypothermia are middle-aged male alcoholics (33,34). Ethanol produces vasodilatation, interfering with the peripheral vasoconstriction that is an important physiologic defense against the cold (7). Although ethanol-containing beverages are sometimes taken in cold surroundings in order to obtain the subjective sense of warmth they produce, this practice is dangerous. Ethanol also predisposes persons to hypothermia indirectly, by inhibiting hepatic gluconeogenesis and causing carbohydrate-depleted persons (like many chronic alcoholics) to have hypoglycemia. Ethanol-induced hypoglycemia has been shown to cause healthy volunteers (35) to have hypoglycemia.

Ironically, ethanol treatment appears to improve survival from a hypothermic episode, an observation that may account for the relatively low mortality observed among alcoholics with hypothermia (36). Ethanol appears to delay the harm produced by impaired circulation and respiration by decreasing cellular metabolism, and thus, the requirement for oxygen—especially in the central nervous system (37). In addition, ethanol may reduce the tendency to ventricular fibrillation in association with hypothermia (36).

Treatment with neuroleptics (e.g., phenothiazines, butyrophenones, and thioxanthenes) also predisposes to hypothermia. Chlorpromazine, the prototype drug of this group, has been used to induce hypothermia pharmacologically (6,38). Chlorpromazine suppresses shivering, probably by a central mechanism, and causes vasodilatation (39). The hypothermic action of drugs of this class becomes more pronounced with decreasing ambient temperature (40).

Other Risk Factors

Infants < 1 year of age have a higher rate of death due to cold than do older children (Figure 2). Neonates, especially premature or small-for-dates babies, are at particularly high risk. Although the mechanisms for maintaining thermal homeostasis (vasoconstriction and thermogenesis by shivering) are present at birth, they seem to function less effectively than in older children. Infants have a relatively large ratio of heat-losing surface to heat-generating volume, and the layer of insulating subcutaneous fat is relatively thin. Perhaps most importantly, a young baby lacks the ability to control his or her own environment. Babies are totally dependent on others to care for their thermal needs, and if sufficient assistance is not forthcoming in a cold environment, hypothermia may result (6).

Hypothermia affecting infants can be a substantial public health problem in areas with severe winter weather. In

December and January of the winters 1961-1962 and 1962-1963, 110 babies with severe hypothermia (temperature < 90 F or 32.2 C) were admitted to hospitals in Glasgow, Scotland. Mortality in this group was 46% (41). Hypothermia affecting babies and young children can also be a winter problem in tropical climates, where it is associated with protein-calorie malnutrition (42).

For older children and young adults, lethal hypothermia is relatively infrequent (Figure 2). However, persons in these age groups are still susceptible to an overwhelming cold stress.

Death due to cold is relatively more frequent among males than females of virtually all age groups (Table 2). The reasons for this are unknown, but differences in the occurrence of risk factors and in patterns of cold exposure between the sexes may play a role.

Hypothermia is common among persons with hypothyroidism. Persons with myxedema (severe hypothyroidism) may be hypothermic without having any unusual cold stress. Lack of thyroid hormone results in a low rate of metabolic heat production, leading to hypothermia (43).

TABLE 2. Death rates* associated with cold (ICD E901), by sex and age and rate ratios, † United States, 1968-1980

COLD (*International Classification of Diseases* E901)

Age	Male	Female	Rate ratio
(in years)			
<1	16.1	9.9	1.6
1	0.9	1.8	0.5
2-4	2.4	2.0	1.2
5-9	0.9	0.3	2.6
10-14	3.4	0.4	8.6
15-24	13.1	3.4	3.8
25-34	17.3	3.3	5.2
35-44	33.1	6.2	5.3
45-54	58.1	14.8	3.9
55-64	86.3	17.6	4.9
65-74	116.8	24.1	4.8
≥75	230.8	73.0	3.2

* Per 10 million population per year.
† Rate ratio = male/female.

Prevention of Illness Resulting from Cold

Since severe illness and death from hypothermia are not only seen in association with cold snaps, efforts to prevent hypothermia must be taken all winter long. Since elderly persons are particularly vulnerable to hypothermia, they form a prime target group toward which preventive efforts should be directed. All dwellings, particularly those in which elderly persons reside, should be properly heated. Local governments can assist in this effort by adopting housing maintenance and occupancy ordinances that require each dwelling to have heating equipment that can safely maintain a reasonable room temperature under expected winter conditions (44). Maintenance of thermal standards is particularly important in nursing homes, hospitals, and other institutions that frequently house the elderly. Economically disadvantaged elderly persons may not make sufficient use of heating equipment because they are unable to pay the resulting fuel bills. In recent years financial assistance has been made available by Federal and certain state authorities to help needy elderly persons pay these bills. Publicity regarding the existence of such programs may enable them to better accomplish their purpose. Health education programs may be useful in informing elderly persons of their susceptibility to the cold.

New parents, pediatricians, and other health professionals involved in the care of babies in their first year of life should be aware that this age group is vulnerable to cold stress. Prevention of hypothermia among infants requires an adequate ambient air temperature and sufficient insulation by blankets or clothing.

Prevention of hypothermia, frostbite, and local nonfreezing cold injury among recreationists participating in winter sports also requires clothing with adequate insulating capacity. Recreationists should make themselves aware of the magnitude of the cold stress likely to be encountered and should dress accordingly. Care should be taken to keep clothing dry and to avoid immersion in cold water (45).

Alcohol and sedative drugs should not be used during periods of cold stress. Persons being maintained on neuroleptic drugs (phenothiazines, butyrophenones, and thioxanthenes) should be advised by their physicians of their increased susceptibility to the cold.

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Floods

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Background and Nature of Floods

Factors That May Contribute to Floods and Flash Floods

Flooding results from a variety of causes. Within a given climatic region, tremendous variations of flooding occur because of fluctuations within the hydrologic cycle and other natural and synthetic conditions. The meteorologic process can be fast or slow and can lead to flash floods, which are an explosive development, or the process can slowly develop into more predictable river and coastal flooding (1).

FLASH FLOODS

The National Weather Service (NWS) has defined flash floods as those that follow within a few hours of heavy or excessive rain, a dam or levee failure, or a sudden release of water impounded by an ice jam. Because of the short warning time involved, the NWS flood forecasting procedures used for large streams cannot respond fast enough. There is also the difficulty of predicting when and where flash-flood-producing rainfall will occur.

Although most flash floods are the result of intense localized thunderstorm activity or slowly moving (nearly stationary) thunderstorms or lines of thunderstorms, some occur in conjunction with tropical cyclones and extratropical cyclones (2).

A number of factors determine why a given volume of precipitation will cause devastation in one area but only negligible damage in another. Forerunners to flash floods usually involve atmospheric conditions that influence the continuation and intensity of rainfall. In the upper reaches of river basins, for example, the flood crest on tributary streams can occur in a matter of hours, or even minutes, from the onset of heavy rain (3). Other factors that can contribute to flash floods include steepness of slopes (mountain terrain), absence of vegetation, lack of soil's infiltration capability, floating debris and ice jams, rapid snowmelt, dam and levee failure, rupture of a glacial lake, and volcanic disturbances (1,2).

It is not surprising to find that the incidence of serious flooding has often shown a marked increase in areas that have been denuded of vegetation. Fast surface runoff occurs on steep slopes and in sites in which the soil's infiltration capacity is reduced. Roofs, pavements, roads, and other solid surfaces have the same effect as an impermeable rock layer, which causes water to move laterally at an increased velocity. Floating debris or ice can accumulate at natural or other obstructions, such as bridges and culverts, creating a dam that may back up flood waters. When a barrier of debris or ice is washed out, a surge wave can create a flash flood.

In mountainous terrain, snow can melt precipitously, resulting in rapid rises in headwaters and downstream rivers. Snow covers can melt explosively in association with rainstorms and a sudden warm spell. Rainfall not only contributes to the volume of the runoff but also helps to thaw the snowpacks (1,4,5).

Dam or levee failure, rupture of a glacial lake, volcanic disturbances, and landslides contribute to flash floods. Lava flows and landslides can cross a stream, creating a temporary dam, which causes flooding upstream and a possible surge of water downstream when the barrier is washed out. The rupture of an ice barrier leading to the sudden release of an impounded lake is an unpredictable natural cause of flooding. Fortunately, most of the world's glacially dammed lakes are located in sparsely settled areas, and most flood-associated events do not approach the magnitude of many human-generated disasters (1,4).

RIVER FLOODS

Factors that influence flash flooding can also contribute to river flooding. Other factors that contribute to more insidious river flooding include stream-channel characteristics, character of soil and subsoil, and degree of synthetic modification in the river regime. Atmospheric conditions that influence the continuation or intensity of rainfall, the amount of precipitation that has fallen into the watershed, snowmelt, the amount of water flowing in tributaries upstream, and the degree of soil saturation also influence the severity of river flooding in an area (1,3).

Urbanization, by increasing runoff and decreasing water infiltration to groundwater storage, changes flooding patterns so that both the height of floods and the areas covered by floods increase locally and downstream. Encroachment on floodplains and valley storage by fillings for buildings,

levees, constructed navigation facilities, and other structures alters the height and duration of river floods. Such alterations to a basin's hydrologic regime increase the risk to inhabitants and structures (1).

Some river flooding may develop principally as a result of snowmelt. The quantity of snow, the rate at which it melts, and ice jams along waterways increase the magnitude of flood hazards. The quicker the thaw, the more dangerous the potential consequences. The state of the ground during the melting phase is also an important contributing factor. Soil percolation is impossible if the ground is frozen to a great depth, and the frozen surface has the same effect as a city's constructed surfaces. Runoff moves rapidly across the surface to the closest stream channel (1,2).

The interactive effects of soil movement, soil temperature, air temperature, and solar radiation on snowmelt are important but poorly understood factors. The time lag between the onset of above-freezing temperatures and melting of snow cover is usually poorly surveyed. The potential for flooding, however, can be determined by examining a wide range of hydrologic and hydraulic variables, including an examination of the water content of the late winter snow cover and the expected precipitation. A spring snowmelt outlook can alert the public to the possible spring flooding danger (1,5).

COASTAL FLOODS

Coastal flooding—defined as including flooding along the Great Lakes—can result from several factors. An important one is storm surge, which is the result of a major tropical storm or hurricane. Winds generated by revolving storms can drive ocean waters inland and lead to serious flooding. A steady buildup of tide level usually occurs; thus, flood warnings can at least alleviate heavy loss of life.

One type of coastal flooding, however, which is often unpredictable, is the most devastating of all. It is called tsunami. This long sea wave is generated by submarine earthquakes or certain volcanic eruptions (see chapter on volcanoes). When the wave reaches shallower water, the tsunami slows down and increases substantially in height; it can destroy coastlines (1,4). Most recorded tsunamis have occurred in the Pacific and Pacific Coast regions. The islands of Hawaii are particularly prone to tsunami damage because of their location in the mid-Pacific.

The degree of flooding is also influenced by land subsidence in coastal areas, erosion of barriers, and the simultaneous occurrence of river floods at a time of a storm surge—or tsunami—or the state of the tide at the time of the peak flood stage. Seiches, which are waves trapped in a basin, occur in large lakes. These can also have an impact on coastal flooding. Damages from coastal flooding, as from river flooding, can be escalated in urban areas (1).

Historical Review of Floods

Populations have been subjected to floods since the advent of civilization. It has been estimated that floods account for 40% of all the world's natural disasters, and they do the greatest amount of damage (6).

FLOODING OUTSIDE THE CONTINENTAL UNITED STATES

Along the Hwang Ho (Yellow) River in China, the most flood-prone river in the world, floods have periodically inundated the land for 40 centuries. The most lethal flood in recorded history struck there in the fall of 1887 when the river overflowed 70-foot-high levees, destroying 11 cities and 300 villages. An estimated 900,000 people were killed and 2 million made homeless. In northern China in 1939, some 500,000 persons drowned in extensive flooding. Several hundred thousand may have died in China's Shantung Province in 1969, when storm surges raked the coastline and pushed flood tides up the Yellow River Valley (6).

A sudden flood in January 1967 in Rio de Janeiro killed 1,500 people. This flood was followed by another on February 20 of the same year, which took 200 lives and rendered 25,000 homeless (7).

In 1974 heavy rains flooded 80% of Bangladesh and caused 2,500 deaths (7).

England and Europe have had their share of floods. The most devastating British inland flood was in August 1952 in North Devon, around the town of Lynmouth, after 9 inches of rain. Little rivers whose water death was usually only a few inches carried torrents of water tens of feet deep. The landscape was vastly altered, 93 buildings were destroyed, and 34 persons lost their lives (6). The Netherlands flood of February 1953, resulting from the breach of a polder, affected extensive areas of the country and caused 1,795 deaths. In October of 1963, heavy rains caused an enormous landslide that fell into the lake behind the Vaiont Dam in northern Italy. Over 100 million tons of water were displaced over the dam top and crashed into the Piave River Valley, almost obliterating the town of Longorone and several nearby hamlets. In Longorone itself, 1,269 of 1,348 people known to be in the town were killed, and an additional 727 persons were killed in nearby locations (6). In 1967, 450 people were killed in a flood in Lisbon, Portugal (7). In 1985 an estimated 7-15 inches of rain fell in a 10-hour period in Puerto Rico, causing 180 fatalities—127 related to a rock slide and 53 related to flooding (L. Sanderson, personal communication).

FLOODING WITHIN THE CONTINENTAL UNITED STATES

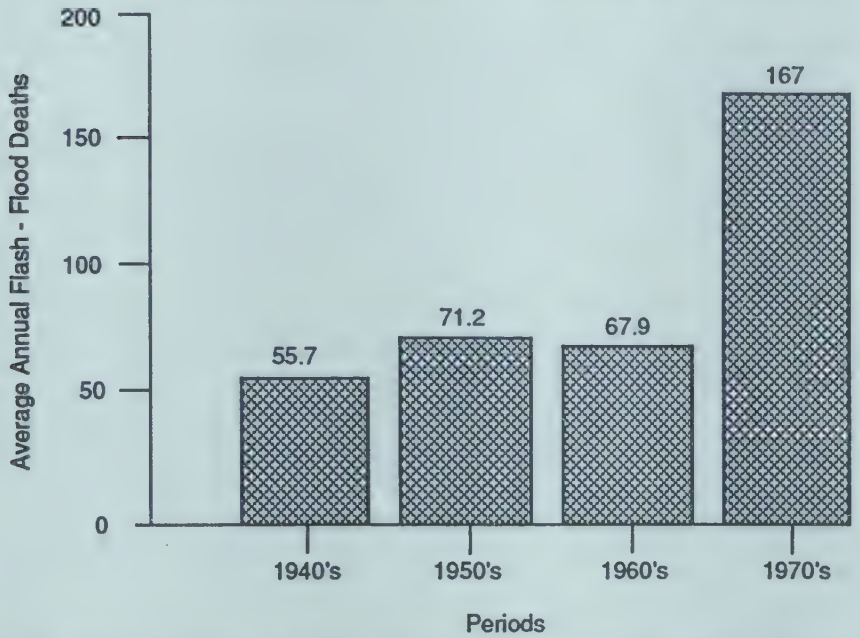
In the United States, nearly every community has some type of a flood problem. Overflowing rivers and streams cause substantial flooding in about half of the communities and over at least 7% of the land area of the nation.

River flooding. One type of flooding is marked by slow, steady rising of the level of a river from melting snow and repeated rainfalls, swelling to its banks, followed by spreading over the land. Rivers turn to seas, houses turn to islands, and crops are buried in mud. In 1913, repeated winter rains caused the Ohio River to overflow its banks, resulting in severe flooding in Ohio and Indiana. Some 730 people lost their lives. In 1927, spring rains caused the Mississippi River to overflow its banks and spread out over 18 million acres of land from southern Illinois to southern Louisiana. All along the river, death came when levees holding back the river broke. A total of 313 persons were killed, and 637,000 were left homeless (6).

Engineering controls—such as the construction of reservoirs and flood walls and the diversion of rivers, as well as increased forestation of watersheds—have reduced river flooding in the past 2 decades.

Flash flooding. Flash floods are distinguished from general river flooding by the very rapid runoff of water after heavy rains. In recent years, flash floods have become the number one weather-related killer in the United States. Deaths from flash floods are approaching 200 each year, compared with an average of less than 70 per year during the preceding 30-year period. Losses from flash floods are now nearly 10 times the levels in the 1940s (8). The average annual number of deaths from flash floods by decade is shown in Figure 1 (2).

FIGURE 1. Average annual flash-flood deaths, by decades*



*Figures are 90% of all flood-related deaths reported by the National Climatic Center, National Oceanic Atmospheric Administration

Source: Reference 2.

The worst flash flood in U.S. history occurred in Johnstown, Pennsylvania, in May 1889 when the South Fork Dam broke after torrential rains, sending the waters of the overflowing Conemaugh Lake Reservoir into the valley below. Among the 2,200 persons who died, 99 entire families were represented. Johnstown experienced another flash flood in 1977 when 8-12 inches of rain fell in the area during the night of July 19-20. Major flash flooding occurred along the Conemaugh River and its tributaries. During the early morning hours, several earthen dams failed. Seventy-six persons were killed, and nearly 2,700 were injured (6).

In 1972, three major flash flood disasters occurred in the United States. Heavy rain accompanied by dam breaks caused flash floods in Rapid City, South Dakota, and Buffalo Creek, West Virginia, causing 236 deaths and 3,000 injuries in Rapid City and 139 deaths in Buffalo Creek. That same year, hurricane Agnes dropped as much as 15 inches of rain in several locations from Virginia to New York. The resultant flooding killed 117 persons and caused damage estimated at $3.1 billion. The entire state of Pennsylvania was declared a disaster area (6).

On the night of July 31, 1976, 10-12 inches of rain fell within a small oval-shaped area over the Front Range of the Rocky Mountains near the head of the Big Thompson Canyon in Colorado. At one location near Glen Comfort, approximately 7.5 inches of rain fell between 7:30 p.m. and 8:40 p.m. An estimated 50 million tons of water were dumped by the rains, mostly in the area between Estes Park and Drake. The canyon was occupied by 2,000-3,000 visitors and summer residents, and 135 people perished in the flood (6).

On September 12, 1977, more than 6 inches of rain fell on Kansas City, Kansas, followed by an additional 5 inches of rain from 7 p.m to 9 p.m. Twenty-five people died in the resulting flash floods (6).

In 1982 an estimated 15 inches of rain fell in southern Connecticut in a 2-day period, resulting in 11 flood-related deaths (9).

In 1985 the heavy rainfall from hurricane Juan, in combination with another tropical depression off the east coast, caused flooding in Louisiana, Alabama, Virginia, West Virginia, and Pennsylvania. In Louisiana, seven people were reported dead and eight missing; the combined flood toll in Virginia, West Virginia, Maryland, and Pennsylvania was reported to be 42 dead and 50 missing (10).

The increased toll from flash floods results from nature, performing as it usually does, colliding with a larger and more urbanized population settling and occupying sites that are ready targets for floods. In the countryside, as evidenced in the Big Thompson Canyon flood, the increased use of mountainous locales and narrow canyons for recreational purposes is exposing growing numbers of unwary visitors to areas susceptible to flash floods.

The list of danger spots is growing. In 1977, more than 15,000 U.S. communities and recreational areas were identified by the Flood Insurance Administration as being susceptible to flash floods. In 1978, the American Meteorological Society urged a concerted effort by Federal, state, and local officials to reduce the losses from flash floods. They called for improved forecasts and warnings, increased regulation of flash-flood-prone areas, certification of dam safety, better information on maximum precipitation and runoff, and programs of public awareness and community warnings (6).

Some evidence suggests that the institution of these measures has decreased the mortality from flash floods. In a review of mortality associated with flash floods from 1969-1981, based on National Weather Service Reports, 2.5 times more deaths occurred in the 14 flash floods in 1969-1976 than in the 18 flash floods in the period 1977-1981. More than twice as many deaths were associated with flash floods for which the survey team considered the warnings inadequate than with those for which the warnings were considered adequate. Warnings deemed inadequate were largely heavy-rain and flash-flood warnings issued for a region within a broad time frame such as the next day rather than for a flash flood in a specific place within a specific, short time frame (11).

Factors Influencing Morbidity and Mortality

Primary Exposures and Effects

ACUTE EFFECTS

Fast-flowing water accompanied by such debris as boulders and fallen trees accounts for the primary flood-related exposure that leads to morbidity and mortality. The way people respond to exposures is a critical factor in the morbidity and mortality associated with such events. The available information on this aspect is limited primarily to anecdotes and descriptive studies and deals only with mortality.

When 7 inches of rain sent flash floods rolling through homes and businesses in Rochester, Minnesota, in July 1978, three elderly patients and a nurse's aide got into an elevator on the first floor to go to a higher floor and escape the rising floodwaters. The floodwaters short-circuited the elevator and instead sent it down into a flooded basement. All four were trapped inside and drowned (6).

Grunfest analyzed the behavior patterns adopted at the time of the Big Thompson Canyon flash flood (12). Comparisons were made of the actions of the survivors and nonsurvivors and the warned and nonwarned populations. Variables were examined such as location before the flood, action taken, grouping of persons, location in the canyon, type of warning received (if any), and number of people in a group. The study sample comprised 54 nonsurvivor groups (129 people) and 58 survivor groups (270 individuals).

Of the 54 groups of people who died, nine groups (17 people) were given an unofficial warning, and five groups (14 people) received an official warning. Persons with recent disaster experience were more likely to take protective action as a result of warnings. An earlier study by Friedsam had shown that older persons were less likely to receive warnings than younger persons regardless of the source (13). However, in the Big Thompson Canyon flood, groups of people > 70 of age were just as likely to receive a warning as younger persons.

Another of Friedsam's finding was that elderly persons were less likely to evacuate than younger persons. This finding was supported in the Big Thompson Canyon study. Of nine groups of people > 70 years of age, three did take action and six took no action during the flood.

Those who took some action were more likely to live than those who took none. The number of people in a group was most significant in characterizing the groups as to whether action was taken. Persons who were alone were most likely to do nothing and had the highest risk of being killed, particularly those driving alone through the canyon. Those who were in groups of three to five friends were more likely to take action, such as climbing the side of the canyon, and they had the best chance to survive. Those who were with family groups were more likely to take no action than they were to climb, but those in family groups were still more likely to live than those who were alone. Those who climbed or took other action were more likely *not* to have received a warning than those who did nothing or who drove.

The location of the group in the canyon was most significant in separating the warned from the unwarned, and those in the upper part of the canyon were more likely to live than those in other areas of the canyon. Familiarity with the canyon itself was not a significant factor in separating the survivors from the nonsurvivors.

Kircher *et al.* studied the factors contributing to the successful evacuation of people in the town of Essex, Connecticut, before five dams were destroyed after heavy rainfall. In that situation, all the people complied with the notice to evacuate even though most of them were not aware that they lived in a flood-risk area. Moreover, most had minimal previous flood experience and did not feel that they were personally at risk during the crisis. The mean age of respondents told to evacuate was 60 years. The authors concluded that in Essex characteristics of trust between town officials and citizens were crucial to a successful evacuation (9).

In the Kansas City, Kansas, flash flood in 1977—in which 25 persons died—a policeman told a Chicago Tribune reporter afterward "A lot of people who died didn't have to die. They just could have walked away from it but they didn't." In most areas the flood was not sudden. No wall of water came crashing down over victims. In the shopping area where many of the dead were found, the water rose rapidly but not so quickly as to explain all the deaths. Anybody who walked to high ground (only two blocks away) could have arrived with little more than wet shoes. However, some people did not walk. Witnesses told of one man who drove up behind a string of stalled cars, some of which were in window-deep water. He pulled around them and drove on into the water. He, his wife, and two children drowned. Some persons ran to save their cars even as other cars floated past them. Others ran into underground parking garages, apparently not stopping to think that these would be among the first places to be flooded (6).

In the study of flash floods based on National Weather Survey Reports for 1969-1981, causes of death were recorded for only 190 cases. Of these 190 deaths, 177 (93%) were because of drowning. A large portion (42%) of the drownings were car-related, such as when victims were in cars driven into low areas, across flooded bridges, or off the road into deep water. The other drownings occurred in homes, at campsites, or when persons were crossing bridges or streams (11).

When hurricane Gilbert struck the Yucatan Peninsula and Northern Mexican Gulf Coast in August 1988 with winds of 160 miles/hour, the greatest loss of life was associated with a single flash-flood event. An estimated 200 people drowned in Monterrey, Mexico, when four buses, evacuating people inland, drove into low-lying areas and were swept away by the fast-rushing water from heavy rain that accompanied the hurricane (14).

The findings from the limited database on the actions taken by people in the face of a flood threat are somewhat inconsistent. In the Big Thompson Canyon study, persons who received warnings—official or unofficial—did not take actions that contributed to higher success of survival than those who had no warnings. These findings concurred with those of Friedsam (13) regarding the reluctance of older people to evacuate or take other appropriate action, but not with those of Kircher *et al.* (9), who found that most people who received a warning in Essex did evacuate and that the evacuees were primarily older people. A possible explanation for these inconsistencies may be the different environmental circumstances surrounding the flood. In Essex members of the volunteer fire department knocked on people's doors to warn them and assisted those who needed help in evacuating. In the Big Thompson Canyon flood, most of the warnings were given by word of mouth, and the most appropriate action for survival was to climb to higher ground, which meant scaling the slippery wall of the canyon.

Taking appropriate action may depend not only on the ability to assess such actions and the willingness to take them but also on having the physical stamina to carry them out. In the analysis of the Big Thompson Canyon study, it was not possible to look at all the group actions by age because of inadequate information. It may be that people

who climbed to higher ground by scaling the canyon wall—particularly the groups comprising three to five friends—were younger and in better physical condition than the other people studied. People in groups of two or more can help each other perform arduous physical tasks compared with those who are alone. Older individuals and families with young children may not be physically capable of climbing to higher ground.

A consistent finding from the anecdotes as well as the more formal studies is the high proportion of car-related drownings in floods. People seem wedded to and unwilling to abandon their cars. Persons trying to leave a flood-threatened area by car may inadvertently drive into a low-lying area or across a flooded bridge. Their cars may stall because of high water or become blocked by rockfalls, mudslides, fallen trees, or other stalled cars. They may find themselves trapped in their cars while high levels of fast-flowing water descend upon them. In many flash floods, the flow of water can be so fast that it leaves very little time for escape.

LONG-TERM EFFECTS

Several investigators have looked at the long-term health effects from floods.

In 1954 Lorraine conducted a mortality study in the 2-month period after floods in the Canary Islands on January 31 and February 1, 1953. Increased mortality was reported for February and March 1953, compared with the same period in 1952. The increased rate was principally among the elderly and those with predisposing respiratory conditions (15).

Bennett studied 316 flood respondents and 454 nonflood controls for morbidity and mortality over the year after the Bristol floods in England in 1968. He found higher mortality among residents of the flooded sections—especially the elderly people. The health status during the year was worse in the flood group when measured by increased hospital admissions for both males and females and a higher surgery rate for flood-group males who continued to live in their own homes. There was also an increase in new psychiatric symptoms for both males and females (16).

Psychiatric examinations performed on 224 children 2 years after the 1972 flood in Buffalo Creek, West Virginia, showed that 80% of the children were severely emotionally impaired by their experiences during and after the flood (17,18).

Melick interviewed 43 "flood" and 48 "nonflood" male respondents for mental and physical problems 3 years after the 1972 flood caused by tropical storm Agnes in Wyoming Valley, Pennsylvania. Flood victims did not report a greater number of illnesses or different types of illnesses from those reported by nonflood victims, but the former group did show longer duration of illnesses. Emotional disturbances occurred in both groups, but they lasted longer for flood-group respondents and their family members (19). Five years after this flood, Logue studied 407 "flood" and 155 "nonflood" female respondents by mailed questionnaire to determine their families' mental and physical health status. Perceived health problems were reported more frequently by flood respondents and their immediate family members. The development of hypertension by the husbands of flood respondents was significantly greater than by the husbands of nonflood respondents in the 5 years after the flood. Flood families also reported more respiratory-, gastrointestinal-, and cardiovascular-related health problems. Flood respondents also reported experiencing more "stress" for major life events after the flood (20). In assessing risk factors for hypertension associated with the early recovery period, Logue and Hansen conducted a case-control study of the 31 female flood victims who reported the development of hypertension in the 5-year post-disaster survey. Factors such as property loss, financial difficulties, physical work, use of alcohol, and perceived distress were significantly associated with hypertension. Anxiety and difficulty in sleeping also demonstrated significant positive correlations with hypertension (21).

Benin reported a marked increase in hypertensive disease in Voroshilovgrad, Russia, after the 1964 flood and in Tiraspol, Moldavia, after two successive floods in 1969. In Tiraspol there was an increase in diabetes as well (7).

During the prolonged flooding of the Amur River in 1951-1952, a strong direct association between flood conditions and blood pressure was reported. Blood pressure readings were taken for the entire population of one village (3,600 people) struck by the flood, and almost everyone showed a general increase in blood pressure. Similar testing was done on the population on the other bank of the river, which was not flooded, and only 20%-22% had elevated blood pressure readings (22).

Abrahams studied 234 flood families and 163 nonflood families by interview 3 and 12 months after the Brisbane floods in Australia in 1974. The number of visits to physicians and hospitals by members of the flood group increased after the flood. Psychological problems were more common than physical problems and were more common for females (23). Price extended the Abrahams study, focusing on age-related health effects. He found that women ≥ 65 years of age experienced significantly more psychiatric symptoms than men. This sex difference was not apparent for persons ≥ 65 years of age (24).

In 1978, Janerich *et al.* investigated a reported cluster of lymphoma and leukemia cases in the Canisteo River Valley in New York (25). Before 1974 the combined rates of leukemia and lymphoma for the river-valley towns and the nonriver-valley towns were similar. In the period 1974-1977, after the severe flooding in the river-valley towns from tropical storm Agnes in 1972, there was a small increase in rates of lymphoma and leukemia in the nonriver-valley towns and a statistically significant increase in rates of these problems in the river-valley towns. The rates for other malignancies were unaffected during this period. The data indicated that the increase in leukemia and lymphoma rates occurred in all three river-valley towns, and the rates for the approximately 100,000 people were about 35% higher than would be expected. Examination of statistics on patterns of reproductivity for the four counties for 1970-1977 showed a statistically significant excess of spontaneous abortions in 1973 compared with average numbers for the other years. Except for 1973, the year after the flood, the spontaneous abortion rates in these four counties were consistently lower than for the rest of upstate New York. The excess was greatest in the river-valley towns.

Environmental monitoring failed to link radiation or poor water quality with the effects observed. The sequence of events in this situation led the authors to identify the flood as a possible causative event contributing to leukemia and

lymphoma for the 2- to 5-year period after the flood and to increased spontaneous abortions for the year after the flood (25).

These follow-up studies show a consistent pattern of increased psychological problems among flood victims for up to 5 years after the flood. The findings regarding nonpsychiatric morbidity are less consistent, but many of the reported morbidity problems such as hypertension and cardiovascular disease—and even leukemia and lymphoma—may be stress related. Greene (26) investigated the relationship of the onset of leukemia and lymphoma to the severe psychological stress by involving feelings of situations loss. At the time of diagnosis, most patients were experiencing a severe psychological sense of loss or hopelessness, which was generated by stress events that occurred as long as 4 years before the appearance of clinical disease. Greene observed that the median interval between the stress event and a confirmed diagnosis was 1 year and that a large cluster occurred 11-13 months before the disease was diagnosed. Neither the short latency of leukemia and lymphoma in the Janerich study nor the prolonged high incidence can be clearly explained, but both phenomena are consistent with Greene's observations about stress events in the prodromal period of adult-onset leukemia and lymphoma.

Secondary Exposures and Effects

Floods may cause disruption of water purification and sewage disposal systems, rupture of underground pipelines and storage tanks, overflowing of toxic waste sites, enhancement of vector-breeding conditions, and dislodgement of chemicals stored above ground. Floods may also lead to overcrowding in temporary shelters. These events may contribute to increased exposure to biological and chemical agents. There is limited documentation of disease outbreaks associated with such exposures after floods.

INFECTIOUS DISEASE

An outbreak of leptospirosis occurred after floods in July 1975 in Greater Recife, Brazil. Of 107 reported cases, 105 were confirmed by seroagglutination or hemoculture. *Leptospira icterohaemorrhagiae* was the predominant serotype found (27).

An increase in malaria was reported after heavy rains associated with a hurricane that struck Haiti in October 1963. The percentage of malaria-positive slides tested rose from 2% in September 1963 to almost 26% by the end of February 1964. The highest positive slide rates were for children ≤ 1 year of age. Both sexes were equally affected. Coastal areas showed higher rates than the interior, and localities at altitudes of < 300 meters showed higher rates than localities at higher altitudes. The investigators attributed the epidemic to an interruption of spraying, which left a large reservoir of gametocyte carriers after the hurricane; a lack of shelter, which resulted in a greater exposure of the population to the principal vector; almost complete removal of the insecticide from homes by heavy rainfall; an explosive increase in mosquito breeding due to heavy rainfall; and increased movement of the population in search of refuge from the storm's devastation (28).

Public health officials of Rybnitzky district in Moldavia reported the number of cases of scarlet fever doubled after the 1969 flood. In Tiraspol the number of such cases tripled, and there were twice as many cases of whooping cough. Increased morbidity affecting children was registered during and immediately after the flood. An epidemic of dysentery also occurred after the 1969 floods in Moldavia (7).

In Voroshilovgrad, Russia, after the flood in 1954, rates of typhoid and paratyphoid were almost twice as high as in 1953. A typhoid epidemic also occurred in Kishinev, Russia, after a June 1969 flood. Typhoid morbidity there was 9.8/100,000 in 1968 and 14.8 in 1969 (7).

A widespread outbreak of yellow fever in Argentina in 1965-1966 was associated with floods that covered the area at that time (7).

Bissel (29) found a significant increase in typhoid, paratyphoid, hepatitis, and measles in the Dominican Republic after flooding caused by hurricanes David and Frederick on August 31 and September 5, 1979, respectively. The author attributed the observed increase in infectious disease to a) flood-caused water transmission of pathogens and b) overcrowding of makeshift refugee centers with insufficient sanitary facilities.

Infectious disease outbreaks in the United States are rare after flooding (30). This may reflect the fact that the reservoir of agents commonly associated with epidemics after floods is relatively low in the United States compared with other parts of the world. However, some potential does exist for waterborne disease transmission of such agents as enterotoxigenic *Escherichia coli, Shigella, Salmonella,* hepatitis A virus, Norwalk virus agents, as well as the agents that cause leptospirosis and tularemia. There may also be a potential for increased risk of transmission of arboviruses due to increased vector populations from flood conditions.

TOXIC EFFECTS

The release of chemicals during floods has been documented, but information on adverse health effects associated with such exposures is limited.

The flood resulting from the collapse of the Teton Dam in southeastern Idaho in 1976 damaged a large area and led to the release of toxicants into the Snake River. At least three commercial facilities containing pesticides were damaged by the flood, and many farm storehouses were also affected. The flood transported much of the pesticide material stored in containers, broke the containers, and dispersed the pesticides over 240 km of the Snake River. A pesticide-recovery team studied the flooded area for 3 weeks and collected 1,104 containers, about 35% of which contained toxicants such as DDT, dieldrin, polychlorinated biphenyls (PCBs), and organophosphates. It was estimated that $< 60\%$ of the lost pesticide containers were recovered. Over 300 samples of fish, plankton, waterfowl, sediments, water, stream drift, aquatic plants, and soil were collected. The levels of PCB and DDT were high in fish and approached the 2,000-μ/kg FDA-proposed tolerance. Very little preflood data on whole fish were available for comparison (31).

Studies have shown that the uptake of a pesticide, such as aldrin ($C_{12}H_8Cl_6$—an aromatic hydrocarbon that affects the central nervous system, liver, kidneys, and skin), is enhanced in rice plants and grains in flooded soils, and the pesticide persists in the plant for a longer period than in nonflooded soils. The aldrin residues dissipated to their half in 90 days in plants from flooded soil compared to 70 days

for plants from nonflooded soil. At the 90-day period, the flooded soil-plant residue of aldrin was converted to dieldrin (32).

Heavy rains and floods caused a break in a pipe at a fertilizer plant in the town of Vila Parisi, Brazil, in 1985, spreading a snow-white cloud of ammonia over the village and causing 63 people to seek treatment for symptoms (33).

The lack of documentation of adverse health effects from flood-associated toxic exposures may be attributable to several factors: a) very few, if any, studies have been done to look for such associations; b) the symptoms associated with toxic exposures are often nonspecific and may have a latency period; this may make it difficult to associate an adverse effect with a toxic exposure and particularly one that is flood-related; and c) in the past there has been little recognition of the potential for release of toxic chemicals during flooding, and such releases have gone unrecognized.

Public Health Implications

Prevention and Control Measures

The principal steps to follow for preventing death and injury associated with floods are:

To identify flood-prone areas and take appropriate preventive action, including the following: the institution of engineering controls such as the construction of reservoirs and flood walls, as well as the diversion of rivers; forestation of watersheds; land-use management to prevent new construction in flood-prone areas and deforestation of watersheds; and increased regulation of flash-flood-prone areas and posting of flash-flood warnings in recreational areas subject to flash floods.

To conduct dam inspections and issue dam-safety certification. An ongoing program of dam inspections—for both public and private dams—should be carried out. Necessary structural improvements should be required at dams deemed unsafe. During periods of heavy rainfall, dams should be monitored for signs of failure, and these observations should be conveyed to public officials responsible for issuing warnings and making the decision to evacuate the population at risk.

To identify meteorologic conditions conducive to heavy precipitation and runoff and to issue forecasts and warnings of floods for a specific geographic area within a specific time frame. The present state of the art in meteorology is very effective in identifying storm systems that may have impact on a region of the country with heavy rainfall and cause flooding. It is less effective in identifying storm systems that may affect a very specific geographic area within a specific time frame and cause flash flooding. The National Weather Service (NWS) tries to compensate for this weakness by using local observers in flash-flood-prone areas who monitor precipitation and take periodic measurements of creeks and rivers during periods of heavy rainfall. These observers have been very helpful to NWS's efforts to provide adequate flash-flood warnings. However, local communities should take responsibility for providing some of this information themselves, since the NWS system cannot cover all potential flash-flood areas.

To ensure that the public is made aware of flood and flash-flood-prone areas and of appropriate action to be taken when the potential for such flooding exists. In situations in which warnings can be issued far in advance of the impending flood, evacuation of the public by vehicle is an appropriate course of action. However, in flash-flood situations in which there may be little time between the issuing of the warning and exposure to high-rising water, it is important that the evacuation route does not require passage through low-lying areas or other danger spots such as bridges. In such circumstances it may be best to abandon one's car to avoid entrapment and to climb to high ground on foot. Special attention should be given to persons who are unable to make such a climb—such as the elderly, young children, and the handicapped. Traveling alone into flash-flood-prone areas may be inadvisable. Persons who are alone during a flash flood are advised to join other people and form a support group. Local officials should be certain that people living in or visiting flash-flood-prone areas are familiar with appropriate escape routes and appropriate actions when faced with a possible flash flood.

The principal steps to follow in preventing morbidity and mortality from secondary exposures relating to floods are given in the sections below.

Infectious agents. In preventing enteric disease transmission when water and sewerage systems have been compromised, it is very important to assure that the water and food supplies are safe to consume and to assure safe disposal of human waste (34,35). When water has been contaminated or is suspected (e.g., by a drop in water pressure, discoloration, turbidity, or unusual odors), of having been contaminated, people should drink only water bottled or trucked from a safe source, water brought to a vigorous boil, or water appropriately disinfected until health authorities indicate that public supplies are again safe. Contaminated food supplies, spoiled foods, and foods potentially hazardous because of interrupted refrigeration should be discarded. Health sanitarians should be consulted about the safety of food, water supplies, and other sanitation issues.

Flood victims and relief workers should always wash their hands with soap and water (boiled or chemically disinfected when no regular safe supply is available) before preparing or eating food, after using the toilet, and after participating in flood clean-up activities.

Mass vaccination programs at the time of natural disasters are counterproductive and divert limited personnel and resources from other relief tasks. Such programs may also create a false sense of security and lead to neglect of basic hygiene. There is usually a public demand for typhoid vaccine and tetanus toxoid after floods, despite the fact that no epidemics of typhoid have occurred after floods in the United States. Additionally, it takes several weeks for antibody to typhoid to develop, and even then, vaccination provides only moderate protection. Mass tetanus vaccination programs are also not indicated. Management of flood-associated wounds should include appropriate evaluation of tetanus immunity (and vaccination, if indicated) as at any other time.

When floods occur in areas with endemic arthropod-borne encephalitides, arthropods known to transmit the disease should be monitored and areas should be sprayed if the vector population increases significantly after the flood.

Adequate interim lodging should be provided for flood victims.

When returning to flooded dwellings, people should pay special attention to structural, electrical, and gas-leak hazards. Additional attention should be given to factors contributing to the growth of fungi and bacteria (adequate clean-up, drying out, and disinfection). Local health agencies and utility companies should be consulted on such matters when indicated.

Toxic agents. Supplies of drinking water and food, such as fish, should be tested for toxic chemical and radioactive agents if there is reason to suspect that such substances may have been released during the flood. Soil surrounding toxic-waste sites and storage lagoons should be inspected to determine whether contamination from overflowing of these storage areas during flooding has occurred. Contaminated soil areas should be declared off-limits to the public until appropriate clean-up has been instituted. The public should be warned not to consume contaminated water or fish if toxic substances are found.

Surveillance

MORBIDITY AND MORTALITY RELATED TO FLOODS

There is a need to assess the trends in morbidity and mortality related to river flooding and flash floods to determine if preventive measures have been effective. The responsibility for these assessments, in general, rests with the local and state health departments. In the past the information on flood-related mortality has been limited to the number of deaths per year, thus making it difficult to determine if a high number of deaths in a given year was the result of a high death toll from a few floods or a small number of deaths from many floods. To properly assess trends, it is important to have a surveillance system that will provide information on the average number of deaths and injuries per flood per year. To accomplish this, systematic reporting is needed whereby all floods and associated deaths and injuries are reported to the National Weather Service. The average number of deaths and injuries per flash flood or river flood per year should then be computed for the United States as a whole and for various regions of the country. The circumstances surrounding death and injury should also be reported.

MORBIDITY AND MORTALITY FROM INFECTIOUS DISEASE

During and after a flood, a surveillance system should be instituted to permit the identification of increased cases of communicable disease in the flood-stricken area. Particular attention should be given to those diseases endemic to the area. Emergency room visits, clinic and physician office visits, and hospital admissions can be assessed for such purposes on a regular and, preferably, daily basis. The data should be analyzed to determine if there was a significant increase in communicable disease in the 2 or 3 months after the flood compared with the 2 or 3 months preceding it. If a significant increase in infectious disease is found, an appropriate nonflood community should be evaluated for the same time period to determine if there is evidence for such an increase in that community.

POTENTIAL EXPOSURE TO TOXIC AGENTS

State and local governments throughout the United States should know exactly where and how toxic materials are stored within the areas of their jurisdiction. This should include materials in underground storage tanks and pipelines, as well as toxic materials stored above ground. Special attention should be given to these storage areas after a flood to determine whether any of these materials have been released into the environment. If release is suspected, environmental monitoring should be carried out to determine contamination of human pathways of exposure. If evidence for potential human exposure is found, the population at risk should be assessed for associated adverse health effects.

Research Recommendations

Although studies conducted during and after floods have provided some information on factors contributing to the risk of morbidity and mortality, some inconsistencies and questions remain that still need to be resolved:

- The factors influencing actions people take in the face of a flash-flood warning and notice to evacuate should be studied further. Attention should be given to the physical stamina such actions require of people of all ages and varying health status.

- Studies should be done to assess the circumstances under which there is sufficient time—after a flash-flood warning is issued—to permit evacuation by car or when it is safer to abandon one's car and escape to higher ground on foot.

- A cohort of flood victims should be followed over time to determine whether they are at higher risk than a comparable group of nonflood victims of having adverse physical and mental health effects.

- Systematic studies should be undertaken to determine whether an increase in certain biological agents results from disrupted water supplies and sewage systems after floods in different regions of the United States. These studies should also look at changes in certain vector populations before and after flooding.

- Systematic studies should be undertaken to look at the release of chemical agents during flooding and the potential for contamination of human pathways from such events.

- A reporting system should be established to more accurately assess the number of deaths and injuries associated with each flood and the circumstances surrounding each flood death and injury.

Summary

It is estimated that floods represent 40% of all the world's natural disasters, and that they do the greatest amount of

damage. In the United States, flooding—marked by the slow, steady rise of the rivers from melting snow and repeated rainfalls—has been curtailed by engineering controls and increased forestation of watersheds. However, flash floods, marked by the very rapid runoff of water after heavy rains, have in recent years become the number one weather-related killer in the United States. The increased toll from flash floods results from natural events that impact on an increased and more urbanized population's settling and occupying sites that are ready targets for flash floods.

The primary cause of death from floods is drowning. Several studies have found that a high percentage of drownings in flash floods are car-related.

A review of NWS reports has shown that more than twice as many deaths have been associated with flash floods for which the warnings were considered inadequate than with those for which warnings were considered adequate. There are conflicting reports regarding factors that influence the actions people take when receiving a flash-flood warning. Some studies have reported that the elderly are less apt to receive warnings and to evacuate when they do receive warnings. Other studies have not confirmed this pattern. Although some studies have reported that people with previous flood experience were more ready to evacuate, another study of a successful evacuation of an entire community found that the population largely comprised older people with no previous flood experience who willingly evacuated even though they did not sense any immediate danger.

The circumstances surrounding the flood and the physical effort required to reach safety may influence whether people will take appropriate action when faced with the threat of a flash flood. In one study, groups of three to five persons were more apt to take appropriate action and survive than persons who were alone.

Countries outside the United States have reported increased cases of infectious diseases such as leptospirosis, malaria, yellow fever, and typhoid fever after floods, but such increases have not been documented in the United States in the past 3 decades.

Release of chemical agents into the environment during flooding has been documented in both the United States and other parts of the world. Data on adverse health effects associated with such exposures are limited but at least one incident has been recorded in which heavy rains and floods caused the break in a pipe at a fertilizer plant, resulting in symptoms in 63 people due to exposure to ammonia. The paucity of data on health effects associated with such exposures may be in part because few studies have been done to look for such associations after flooding.

Follow-up studies of flood victims show a consistent pattern of increased psychological problems affecting flood victims for up to 5 years after the flood. The findings regarding nonpsychiatric morbidity are less consistent, but chronic effects such as hypertension, cardiovascular disease, leukemia, and lymphoma have been found significantly elevated among flood victims compared with controls in several studies. These observations, however, need confirmation.

Prevention of death and injury from floods can be accomplished by identifying flood-prone areas and taking appropriate preventive action, conducting dam inspections and issuing dam-safety certification, identifying meteorologic conditions that will contribute to heavy precipitation and runoff and issuing warnings of floods for a specific geographic area within a specific time frame, and making the public aware of flood- and flash-flood-prone areas and advising them on appropriate actions to take when faced with a potential flash flood.

Morbidity and mortality from secondary exposures related to floods can be prevented by assuring that water and food supplies are safe to consume and are not contaminated with biological and chemical agents, and by instituting safe human-waste-disposal practices. Soil surrounding toxic waste sites and storage lagoons should be inspected to determine if there has been contamination from overflowing of these storage areas during flooding. Contaminated soil areas should be declared off limits to the public until appropriate clean-up has been completed. Consideration should also be given to potential increases in certain vector populations. Mass vaccination programs at the time of natural disasters are counterproductive.

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Famines

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Introduction

The recurrent specter of famine provides one of the most dramatic examples of contradiction in our modern, technologically advanced world; however, it is far from being a contemporary phenomenon. Historically, famine dates back to Biblical times, with the earliest record of famine—found at the First Cataract of the Nile—more than 5,000 years old (1). References in the Old Testament to 7 years of famine (Genesis 47:13-26) probably date back to around 1,700 B.C. (1). Most recently, a severe famine ravaged many parts of sub-Saharan Africa in 1984-1985, which may have killed more than a million people. In addition to high mortality, the consequences of famine can be far-reaching, creating mass migration of the affected populations. More than a million Irish escaped the potato famine of 1846-1850 and emigrated to America. Members of other populations have crossed into neighboring countries in large numbers, such as when the Kampucheans moved into Thailand in 1979, and the Ethiopians fled into Somalia in 1979-1980 and Sudan in 1984-1985.

Some of the most serious famines of the 20th century have occurred in Europe (e.g., the Ukraine in 1921 and Holland in 1944-1945); however, since the end of World War II, famine has exclusively been a problem in certain areas of the developing world. Hardest hit regions have included the Sahel and the Horn of Africa, and southern and eastern Asia. Economic development, improved agricultural techniques (e.g., the green revolution), improved information dissemination, government systems of food procurement and distribution, and more equitable social systems have all but eliminated famine in developed and many developing countries. Table 1 lists some of the most severe famines of this century.

Definition and Causes of Famine

Famine is defined as a condition of populations in which a substantial increase in deaths is associated with inadequate food consumption (2). The key word in this definition is consumption, since—contrary to popular belief—famines are not always due to problems of food availability. While natural catastrophes often act as a trigger, the underlying conditions within a population which allow famine to develop are generally human generated. Lack of food for consumption may be due to one or both of the following events:

1. Failure to produce food, because of adverse climatic or other environmental conditions. Contributing events may be slow in onset, such as drought or locust infestation, or acute, such as severe flooding, which has often been a precipitating factor in Bangladesh.

2. Failure to distribute food and/or collapse in the marketing system affecting part or all of a population due to political, environmental, or economic crises.

Famine is usually the escalation of an already-existing situation of high undernutrition prevalence in which many individuals within a population experience starvation during so-called "normal" times. Such situations are characterized by endemic poverty, landlessness, intractable debt, and underemployment. The mechanism by which labor, services, or goods are exchanged in normal times for food has been termed "entitlement" by Sen (3); when this process fails the onset of starvation can be rapid. Frequent crop failures in Ethiopia, Somalia, and the Sahel in recent years have been attributed to progressive deterioration of the ecologic environment. Rapid desertification and extensive soil erosion have resulted from deforestation, poor agricultural practices, and over-grazing by pastoralists (4); however, all these environmental factors have been strongly influenced by social and political forces. Famine generally reflects profound societal ills.

Loss of purchasing power through rapid inflation and/or unemployment—such as occurred in Ghana in the late 1970s—may lead to a collapse of the market system. Hoarding, imagined scarcity, and price manipulation were major causes contributing to the 1943-1944 famine in Bengal in which an estimated 1 million people died (3,5).

A common feature of many recent famines has been the influence of widespread political upheaval and armed con-

TABLE 1. Famines of the 20th century

China	1920 and 1960
Germany	1917-18
Russia	1919
Ukraine	1921
India (W. Bengal)	1943
Holland	1944-45
Ethiopia	1972-74
	1984-85
Bangladesh	1971
	1974-75
Sahel	
Senegal	1968-73
Mali	1985
Upper Volta	
Niger	
Chad	
Nigeria (Biafra)	1969
East Timor	1976
Kampuchea	1979
Uganda	1981
Sudan	1985

flict. During the Nigerian civil war, the war which resulted in the creation of Bangladesh, the chaos of the Khmer Rouge regime of Kampuchea, and the disturbances preceding the overthrow of Haile Selassie in Ethiopia, famine occurred among civilian populations. In the 1980s, armed secessionist movements in the northern Ethiopian provinces of Eritrea and Tigre and in the eastern region of the Ogaden, and widespread antigovernment insurgency in many parts of Mozambique have been associated with severe famine, resulting in the deaths of hundreds of thousands in each country (6,7). Not only does this upheaval contribute directly to the creation of a famine situation, but it also seriously disrupts famine relief operations.

Most disturbingly, intentional starvation has apparently been used as a deliberate weapon by some governments and political organizations. Examples of such include the allied blockade of Germany in 1916-1918 (8), the siege of Leningrad in 1941, the war between federal Nigerian forces and the secessionist state of Biafra (9), and the obstruction of relief aid to the Ethiopian provinces of Eritrea and Tigre in 1984-1985 (10) and again in late 1987 (11).

Although historians, disaster-relief organizations, and the press frequently attribute a famine to a single cause (e.g., the Irish potato famine, the Sahelian drought), the cumulative and frequently synergistic interactions of multiple risk factors upon a population are often responsible. The following description of famine in Karamoja, Uganda, in which infant mortality was estimated >600/1,000 live births, provides an example (12):

> With attention focused on political problems in the aftermath of Uganda's liberation, drought in remote Karamoja went unnoticed. Crop failure has a long history and is substantiated by agricultural records since 1924. But the Karamojong have always had a good security system which allowed several fall-back positions, thus avoiding full-scale famine during years of crop failure: reliance on blood and milk from their livestock, the trade of cattle to neighboring districts for grain, interval commerce of surplus grain and food distribution through local government administration.

Famine in Karamoja resulted not only from the drought but also from the breakdown of the reserve food-supply system. Alertness to the multiple risk factors that may lead to famine is essential to the early recognition of prefamine situations and to the initiation of corrective action. The following list categorizes these risk factors by time, place, and person (13).

TIME
- Seasonality of food availability and malnutrition
- Triggering event (natural disaster, war, crop failure)

PLACE
- Political instability
- Developing, poor societies
- Marginal lands (deforested lands, steep slopes, eroded land, desert, low-lying delta)
- Agrarian or nomadic, pastoralist societies (dependent upon single crop or livestock)
- Fragile food chain: production, distribution
- Weak information dissemination and transportation infrastructure
- Limited governmental capacity to predict and respond to food shortages

PERSON
- Infants, young children, elderly people, pregnant and lactating women
- Poor, landless, underemployed, and unemployed families
- Ethnic, religious, and political minorities
- Refugees and internally displaced persons
- Chronically ill and undernourished individuals with restricted nutritional reserve

Detection of Famines

Despite modern systems of information dissemination, the recognition of—and response to—famines in the 1980s has been slow. Even at times that objective data have indicated a serious and worsening situation, such as in Ethiopia in 1984, major food mobilization did not occur until international public opinion was aroused through massive and graphic media depictions of starving and dying children (14-16). Thus, the prevention of famine requires not only a sensitive and specific warning system but also—and above all—an increased willingness and capability of governments and international relief-assistance organizations to respond quickly to prefamine situations.

Traditionally, famines have been assessed in terms of cases, rates or degrees of malnutrition, and/or numbers of deaths from undernutrition. Such indicators quantify the damage—much of it irreparable—that has taken place, but they are of limited use in terms of prediction and prevention. Improved famine detection requires that authorities responsible for famine response shift their attention from the traditional "trailing" indicators to more appropriate "leading" and perhaps "intermediate" indicators, as shown below.

LEADING INDICATORS
- Low acreage under cultivation
- Drought
- Floods
- Low food reserve
- Political instability
- Population movement
- Strong black market
- Insect infestation (e.g., locusts)

INTERMEDIATE INDICATORS
- Crop failure
- Increased price of staples
- Rise in ratio of staple crop price to daily wage
- Increased lending rates
- Sale/consumption of livestock
- Death of livestock in pastoral societies
- Sale of valuable possessions (animals, jewelry, ornaments, farm tools) at less than market value
- Increased seed cost
- Seed shortage
- Consumption of seed grain
- Sale of land
- Population migration
- External market-price manipulation

TRAILING INDICATORS
- Increased rates of low or abnormal anthropometry
- Edema/marasmus among young children
- Increased rates of vitamin deficiencies
- Increased rates of other nutritional deficiencies
- Increased mortality

If trailing indicators alone are relied on to trigger a response, a portion of the high-risk population will experience excess morbidity and mortality. In the words of Jon Rohde (13), "The earlier the intervention, the more likely the later manifestations (particularly deaths) will be avoided. When market prices rise, government grain release can assure stable affordable supplies. When buying power is reduced, food-for-work or test relief projects can provide needed purchasing power." A word of caution here regarding government subsidies of food prices for certain segments of the population, such as urban dwellers, since this may act as a disincentive for farmers and may lead to decreased food production.

New satellite technologies are particularly applicable to improved monitoring of some leading indicators. Through the use of the National Oceanic and Atmospheric Administration satellites' intensified imagery and computer analysis, historical weather records, and crop condition indices, drought and food shortage alerts on some 400 agroclimatic zones are available biweekly from the Assessment Information Service Center. This system, in its fourth year of operation at the time this report was written, is expected to provide a 3- to 6-month lead time on potential drought and food shortage areas (17).

Currently, the U.S. Agency for International Development is implementing a famine early-warning system in sub-Saharan Africa. Available local data, reports from satellites, and selective field surveys are being used to track changes in the level of critical risk factors. This new system might substantially increase the timeliness of recognition and response to famine (18); however, it is one thing for technicians to provide data that indicate the imminent onset of famine and quite another for politicians and decision makers to trigger an adequate response.

Effective early-warning systems would have the distinct advantage of enabling governmental and other types of organizations to provide food to people who need it, without disrupting the recipients' social structures. Once famine and migration occur, people move to urban slums or temporary settlements or "camps" in which population density is high, housing poor, sanitation almost nonexistent, and health services are lacking. Crowding itself leads to an increased risk of exposure to infections. The recognition of impending famine and the provision of food to people in their normal surroundings diminish social disruption, limit exposure to infectious agents, minimize the hopelessness of dependency, and increase the probability of eventual rehabilitation.

Health Consequences of Famine

Just as the causal structure of famine is multi-factorial, the health consequences of famine emerge from multiple aspects of the famine phenomenon. The migration of people away from their homes, their congregation in crowded and unsanitary camps, the frequently associated political violence, and the insidious depression and apathy that result from their plight all contribute to the health problems that afflict victims of famine.

The most obvious results of famine are severe undernutrition and death. While studies have demonstrated that undernourished individuals—especially young children—are at higher risk of mortality (19), the immediate cause of death is usually infectious disease. The groups within a population that are most vulnerable to the effects of famine are also those that are at high risk of premature mortality in non-famine times—namely the poor, the landless, the underemployed, and—within these groups—the oldest and youngest.

Protein-Energy Malnutrition

The signs and symptoms of protein-energy malnutrition (PEM) have been adequately described elsewhere (20,21). In famine situations, the victims of starvation undergo devastating physical and psychological deterioration. Starving individuals generally show weight loss, weakness, apathy, and depression, and symptoms progress to cachexia, diarrhea, anorexia, immobility, and finally death. Edema is rarely seen in association with total starvation but is commonly seen with semi-starvation (8). While the predominant type of PEM affecting young children is marasmus, there are circumstances that can produce kwashiorkor, such as those in Biafra in 1969 when children were given cassava as an energy source but received almost no protein (9).

DIAGNOSIS

Reflecting the situation in "normal" times, PEM affects certain segments of the population more than others. Most severely affected are persons with the highest energy and protein requirements—infants, young children, pregnant and lactating women, the chronically ill, and the elderly. Nutrition status is measured for individual children by anthropometry, whereby certain measurements are compared with those for the World Health Organization (WHO) reference population. The most commonly used indices are weight-for-height, weight-for-age, height-for-age, and mid-upper arm circumference (MUAC).

The index that most sensitively detects recent PEM, or wasting, is weight-for-height (22), which is used to assess children <5 years of age or <115 cm in height. MUAC—provided it is accurately measured—can be useful to screen children 1-5 years of age. Assessment of PEM for older children and adults is done by clinical examination. Height-for-age is a sensitive index for assessing long-term PEM, or stunting, and weight-for-age reflects either recent or longer-term nutrition status.

Acute PEM is generally defined as weight-for-height >2 standard deviations below that for the median reference population, or mid-upper-arm circumference of <13.5 cm. Severe PEM is usually defined as weight-for-height >3 standard deviations below that for the median reference population, or a MUAC of <12.5 cm. In the past, moderate and severe PEM have been defined as weight-for-height <80% or <70%, respectively, that of the median reference population.

PREVALENCE

The prevalence of PEM in populations affected by famine has only been estimated by anthropometry within the past 20 years. Prevalence rates as high as 70% were recorded for children in Ethiopian camps in 1985 (23). Estimates have been made through random sampling of populations, screening total populations as they arrived at settlements, and as they participated in mass public health campaigns such as vaccination. Table 2 lists the prevalence of PEM recorded for various famine populations since 1975.

Many, though not all, nutrition surveys have been performed in relief settlements or refugee camps in which the most severely affected elements of a population may have gathered or in which nutrition status may have deteriorated because of inadequate rations and frequent infections.

Micronutrient Deficiencies

Although not commonly perceived as important by the public or by medical-relief workers, several of the micronutrient deficiency syndromes have contributed greatly to mortality and disability among members of populations with acute starvation. Xerophthalmia related to vitamin A deficiency was first described in the Irish famine of 1845-1847 (31). Numerous reports of vitamin A-related blindness among famine victims have since been published (32). In addition to being an effective mechanism for preventing blindness, vitamin A supplementation has recently been suggested as having a role in the prevention of mortality among children (33).

Vitamin C deficiency (scurvy) has been reported from refugee populations in both Sudan and Somalia (23,34) with thousands of individuals being affected. These outbreaks have occurred when traditional diets containing vitamin C (e.g., camel's milk in the Ogaden) were replaced by imported rations of cereals and oil that contained no vitamin C. Likewise, such rations often have not contained enough iron and folic acid for the needs of women of child-bearing age, and widespread iron- and folate-deficiency anemia has been reported in association with famine and refugee situations (35).

Infectious Disease

Most famine-associated deaths result from infection. There are two reasons for this. First, the poor environmental conditions in relief settlements promote the spread of infectious disease. Second, the body's immune defenses against these infectious diseases are seriously impaired as a result of starvation.

In such settings, one of the most important infectious diseases is measles, which causes high mortality rates among young, undernourished children. Reports from the refugee camps of eastern Sudan attribute 50% of all deaths in February 1985 to measles, describing measles-specific mortality rates of 30/1,000/month for children <5 years of age and case-fatality rates as high as 32% among infected children in one camp (29,36). Measles case-fatality rates of 50% were

TABLE 2. Undernutrition prevalence in famine-affected populations

Population*	Year	Percent acute undernutrition†	Reference
Kampuchean refugees	1979	10-18	24
Ogaden refugees in Somalia	1980	28-39	25
Mozambique	1983	12-28	7
Mauritania	1983	8.2-17.2	26
Niger	1984	11.5-12.2	27
Burkina Faso	1985	6-10	28
Tigrean refugees in Sudan	1985	13.8-50	29
Eastern Sudan (nonrefugees)	1985	17	29
Ethiopia (Korem camp)	1985	70	23
Ethiopia (N. Shoa)	1985	26	30

*Based on surveys of children < 5 years of age.

†Defined as weight-for-height < 80% median World Health Organization reference population.

described among children with kwashiorkor during the Nigerian civil war (37).

Diarrheal diseases are common—especially in camps in which the water supply is unsafe and sanitation facilities are absent. While acute, watery diarrhea accounts for most of the morbidity and mortality in these situations, large outbreaks of cholera and bacillary dysentery have been reported from Sudan, Somalia, and Ethiopia (36,38). Other communicable diseases that often afflict famine-affected populations include acute respiratory infections, meningitis, hepatitis, tuberculosis, typhus, relapsing fever, and typhoid fever. It should be stressed, however, that significant mortality from epidemics of unusual diseases has been the exception; the rule has been that *people have most often died from such common diseases as measles, diarrhea, and pneumonia* (23,36).

Problems of Food Toxicity

When common foodstuffs are scarce, there is increased consumption of wild, or so-called "famine foods". Foods that were previously not eaten or else would normally be prepared differently may be consumed in desperation. Reports from Mozambique in 1983-1984 (39) describe many deaths among starving villagers who ate parts of the cassava plant that contain cyanogens that break down into cyanide. This event occurred because normal precautions against toxicity were not taken during these times of extreme hunger.

Mortality

For an individual with severe protein-energy malnutrition and infection, death is the inevitable end result. In a famine setting, large numbers of deaths occur; however, accurate mortality figures are often lacking and difficult to obtain. Mortality estimates that are prepared most commonly come from camps or settlements.

Point estimates of mortality in certain camps have been so high that if translated from daily to annual mortality rates they would approximate 100%. For example, in one camp in Ethiopia, the average daily mortality rate between October 1984 and January 1985 was 49/10,000/day (23). In the Wad Kowli camp of eastern Sudan, mortality rates of 30/10,000/day were recorded in February 1985 (23).

In one of the few population-based mortality surveys in Ethiopia in 1985, a crude mortality rate equivalent to an annual rate of 92/1,000 was found (30). Overall, a conservative estimate of excess mortality (i.e., the mortality over and above that recorded in non-famine times) in 1985 from the Ethiopian famine has been put at 50/1,000. This represents approximately 500,000 deaths in the affected regions of the country. In the eastern Sudan camps, an estimated 5% of the people in eight Tigrean refugee camps died in the first 3 months the camps operated (36).

Age-specific mortality data from Ethiopia and Sudan in 1985 showed that children 1-4 years of age suffered the greatest *excess* mortality of any age group (23). During the 1971-1972 Bangladesh war, however, the highest excess mortality (expressed as percentage increase over normal mortality rates) was found for children 5-9 years of age (40). Infants <1 year of age often experience the highest *absolute* mortality rates of any age group (36); however, the increase over "normal" infant mortality rates is not as significant as that for older children. Surveys in Darfur, western Sudan, in 1985 showed that excess mortality was far higher for boys 5-9 years of age than for girls in the same age group. No such sex differential was found for mortality for other age groups (41).

Different mortality rates have been identified within certain socio-economic groups, even when the difference in resources available to the groups seems minimal. For example, in a rural area of the Bangladesh delta during the post-flood famine of 1974, the mortality rate for landless peasant children 1-4 years of age was 86.5/1,000, while mortality for children in families that owned at least 3 acres was 17.5/1,000 (40).

Response to Famines

A food shortage should be responded to long before undernutrition prevalence increases in a population (see the section on prevention of famines). Even when it appears that an overall food deficit that cannot be prevented by local redistribution of resources is going to occur, timely intervention with food supplies from non-local sources should still be possible. Such intervention depends largely on the quality of the surveillance system in place and the willingness of donors to respond promptly. Given the abundant food reserves in Western countries and even in a number of developing countries (e.g., Zimbabwe, Thailand), there is no logical reason to prevent organization of effective relief programs. Relief operations face a number of constraints locally—including lack of transport and fuel; poor information dissemination; lack of available, skilled managers; and armed conflict in the areas of greatest need. A relief program requires good management, logistics, and nutrition expertise. Medical personnel should play a complementary role. Of profound importance is leadership; one of the major problems in a relief program is the multiplicity of agencies; thus, it is essential that a **lead** relief agency be designated which is either part—or has the active approval—of the government of the affected country.

There are basically four main components of a famine relief program—feeding, prevention of disease, treatment for disease, and epidemiologic surveillance. Feeding is the critical component.

Feeding

Undernourished populations are at an increased risk of dying. A comparative study of the Kampuchean refugee population in Thailand in 1979-1980 and the Tigrean refugee population in Sudan in 1984-1985 clearly demonstrated the close association between undernutrition and mortality in such displaced, famine-affected populations. While each group had a high prevalence of undernutrition upon arrival at the various camps, the mortality rate for the Kampuchean population fell rapidly as the undernutrition prevalence decreased. The opposite was the case for the Tigrean population, for which both the undernutrition prevalence and mortality rate remained high for 7-8 months (29).

One critical difference between the relief operations for the two populations was the numbers of calories provided daily to refugees in the early phases. Simply stated, insufficient food was provided to the Tigrean refugees. Distribution of adequate food to all is essential for the nutritional rehabili-

tation of undernourished populations and cannot be replaced by either selective feeding programs or other preventive programs. Four food-distribution strategies have been described (42).

General food distribution. Dry food is distributed to people who are able to prepare their own meals—preferably in their own homes.

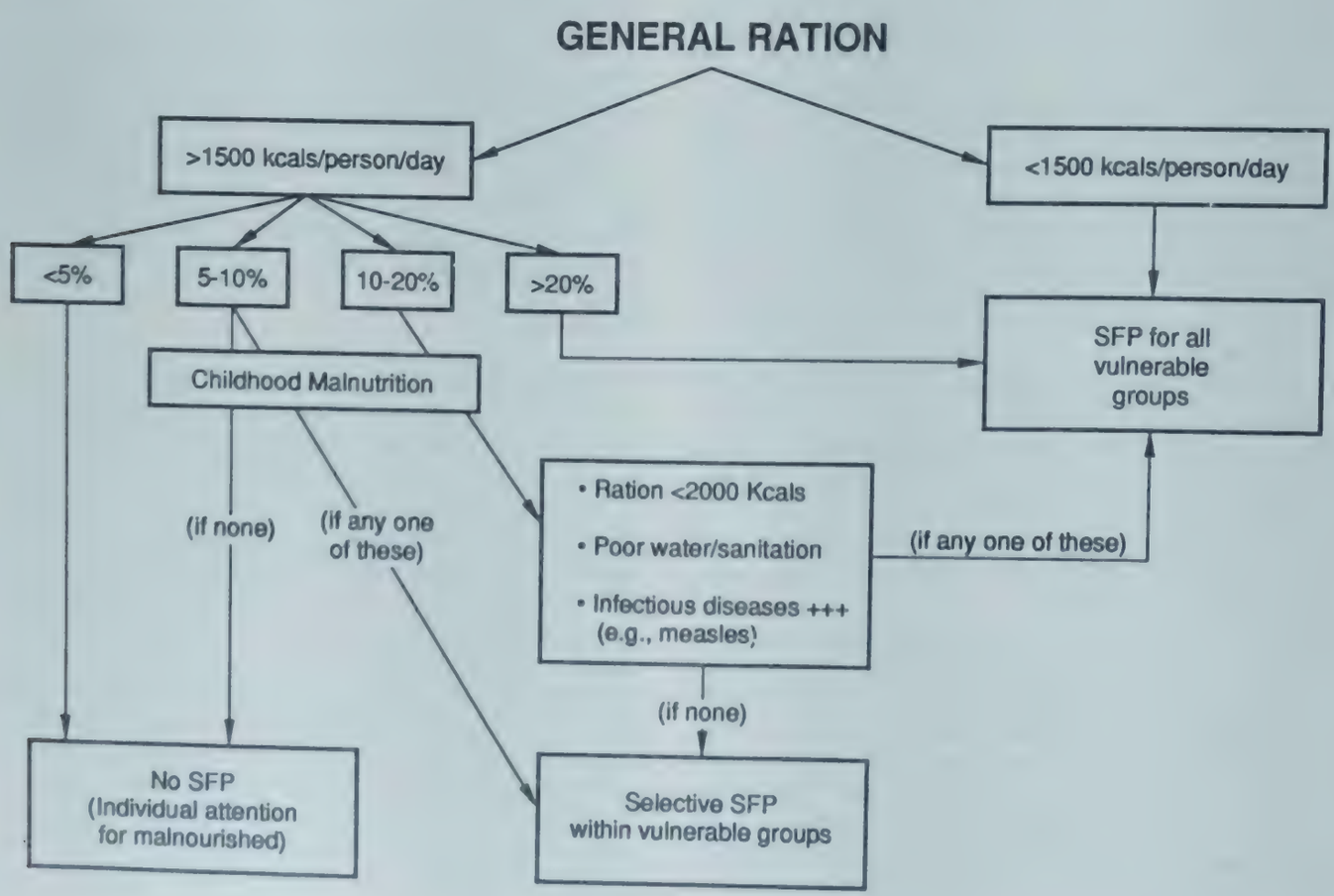
Mass feeding. Prepared, "wet" meals from a central kitchen are served to the population, usually in a camp setting.

Supplementary feeding. In addition to the ration described above (dry food or wet meals) for the whole family, vulnerable groups receive an extra meal or ration to meet their particular needs. This is basically a preventive feeding program for those at high risk.

Therapeutic feeding. Special high-energy, high-protein foods are fed to persons with protein-energy malnutrition. This is a curative program. The procedures for distributing food and organizing various feeding programs are described in several excellent manuals produced by the WHO and Oxfam (42,43). Certain important principles which should be followed in a famine relief operation are listed below.

- Every attempt should be made to distribute food in a community setting, rather than attracting people to relief camps where infectious disease will be endemic and mortality will increase.
- So-called "survival" rations (1,500-1,800 kilocalories/person/day) should not be relied upon for the nutritional rehabilitation of undernourished populations. These rations may allow the well-nourished to survive short periods of time; however, famine victims are generally already suffering from prolonged undernutrition by the time a relief program commences and need a full ration for survival. As a guide, a daily ration containing at least 1,900 kilocalories should be provided to each person, regardless of age.
- While satisfactory energy intake is the most important component of the diet, foods containing protein, iron, vitamin A, vitamin C, and other essential nutrients must be included in the general ration in adequate quantities. Vitamin A supplementation is recommended for all children >6 years of age in famine situations in developing countries (44). Iron supplements are also recommended for pregnant and lactating women (45).
- Supplementary feeding programs (SFPs) are only of value if a full general ration is being distributed to all family members. SFPs are often poorly attended for a variety of reasons (46). These include the inability of mothers to accompany children in feeding centers for long periods of time when they have to care for other sick siblings, collect water and cooking fuel, and prepare meals. Full enrollment of eligible children in SFPs will only occur if the community is actively searched, if the community is educated to the benefits of the program, and if population-based surveys monitor its coverage. Several thorough reviews of SFPs exist in the literature (47,48).
- Deciding whether to begin an SFP in a particular population depends on a number of factors. While there can be no rigid rules for making such a decision, Figure 1

FIGURE 1. The indications for implementation of supplementary feeding programs in famine relief



presents an algorithm that might assist the decision-making process. It is based on the nutritional status of children in the population, the adequacy of general rations, and the presence of such communicable diseases as measles. The algorithm is adapted from Oxfam's *Practical Guide to Refugee Health Care* (49).

- General rations should routinely include items appropriate for use as weaning foods in order to prevent weaning-age children from becoming malnourished in the relief-program environment (50). Utmost care must also be taken to provide grinding facilities for unmilled grains, sufficient cooking fuel, and community education on preparing unfamiliar foods.
- Many imported food items may be too unfamiliar or culturally unacceptable (e.g., some canned meats) to be of value in a relief-assistance program. Problems are frequently encountered with donated dried milks, dried eggs, dairy products, and canned baby foods. A mechanism should be in place to screen unsolicited donations of food (and non-food) items and reject them if they are not useful.

Prevention of Disease

Measles vaccination is the single most important and effective preventive measure that can be taken during a famine emergency. Within the crowded environment of many famine situations, measles—once introduced—spreads explosively and is associated with high mortality. Measles vaccination is best administered to children upon their arrival at a camp or at the beginning of a village-based relief program. It can be combined with initial nutritional screening and other preventive activities such as vitamin A supplementation.

Although WHO recommendations for routine measles vaccination target the 9- to 23-month age group, the increased mortality risk associated with measles in undernourished populations warrants an expansion of the age for measles vaccination to 6-60 months. Undernourished children respond serologically to measles vaccine, and there is evidence that it is protective for most of them (51). Some 30%-50% of children given measles vaccine at 6-8 months of age will not respond immunologically because of residual maternal antibody; thus, children vaccinated before 9 months of age should be revaccinated at 10 months (52). While measles vaccination is an absolute first priority, other vaccinations (diphtheria-pertussis-tetanus, poliomyelitis) should follow when adequate food, water, and sanitation have been provided.

Another major cause of famine-associated mortality is diarrheal disease. Although not all diarrheal diseases are spread by the fecal-oral route, the provision of safe drinking water and sufficient quantities of water for domestic purposes, simple—but well-maintained—sanitation facilities, appropriate weaning foods, encouragement of breast-feeding, and a hygiene education program will minimize the enteric disease risk. The Office of the United Nations High Commissioner for Refugees estimates that at least 15-20 liters of water are required/person/day in a camp situation (45). Expert assistance is needed to properly plan water and sanitation systems. However, careful consideration of water and drainage needs in the selection of camp sites can prevent many unnecessary problems.

Treatment for Disease

Since the response to famine frequently mobilizes medical personnel from a variety of backgrounds, both the establishment of a health-coordination unit and the development of standard criteria for diagnosis and treatment for common conditions are essential. The selection of treatment regimens requires understanding of endemic diseases, drug availability, drug efficacy, costs, and local practices.

An important principle in the organization of curative services in a famine-relief situation is decentralization. Large, crowded outpatient and inpatient facilities create chaos, promote the spread of disease, and take up a disproportionate amount of medical personnel time. Prompt training of health workers selected by the local community in the prevention and treatment of common ailments—as well as the establishment of small, peripheral health posts—can expedite adequate coverage of the population with curative services. Inpatient facilities should be kept to a minimum.

Two major causes of morbidity (and mortality) in undernourished populations are diarrhea and acute respiratory infections. Some 30%-40% of famine-related deaths are associated with diarrhea (36). Although diarrhea can be of noninfectious (nutritional), viral (measles, rotavirus), or bacterial *(Escherichia coli; Shigella, Vibrio cholerae)* origin, appropriate clinical management can significantly reduce mortality from dehydration. The WHO manual titled *The Treatment of Acute Diarrhea* (53) provides guidelines on the use of oral rehydration therapy, intravenous fluids, and antibiotics. Guidelines for the treatment of acute respiratory infections are also available from WHO (54).

Several manuals describe the management of common health problems in famine and relief-camp situations (45,55).

Epidemiologic Surveillance

Epidemiologic surveillance provides quantitative data on populations at risk, identifies health problems that require special action, and monitors the effectiveness of input directed at reducing mortality and improving nutritional status. During the Nigerian civil war, famine surveillance was divided into three stages by Foege (56): immediate ('quick and dirty'), short-term, and ongoing.

IMMEDIATE SURVEILLANCE

Immediate surveillance consists of a rapid appraisal of an affected area, the population involved, and the nature and extent of the problem. After the 1970 cyclone in East Bengal, the use of helicopters enabled authorities to rapidly identify relief needs. In that disaster, which caused an estimated 225,000 deaths, the initial global response was to provide medical relief teams and field hospitals. However, the helicopter survey of the affected area revealed high mortality and few injuries, but a complete loss of seed and draft animals. Relief response, therefore, was shifted from the provision of medical care to the provision of seed and

animals. An in-depth, follow-up survey confirmed the major findings of the rapid assessment (57).

Another example of rapid assessment was in the Sakeo refugee camp in Thailand. A 1-week survey of 33,000 newly arrived Kampuchean refugees identified high mortality—9.1 deaths/10,000 population/day, equivalent to an annual rate of 332/1,000 population—and an unexpected problem of severe malaria (58).

SHORT-TERM ASSESSMENT

Short-term assessment provides two types of data—a cross-sectional assessment of the health status of the population and the identification of high-risk groups for special feeding programs.

Mortality data are basic to this assessment, since they provide a clear, easily recognizable measure of the severity of the situation and establish a baseline for measuring trends over time. Data on deaths, however, are not always easy to obtain. If all deaths occur in medical facilities and are registered, or if registration is required before burial, mortality can be measured or tracked through the use of available data. Otherwise, creative approaches are required, such as monitoring fresh graves, counting the burial shrouds distributed, or reviewing vouchers of burial contractors.

Although mortality rates are generally expressed as deaths/thousand/year, during famine situations they are better expressed in terms of deaths/10,000 persons/day or/1,000 persons/month. This facilitates the monitoring of mortality trends over short periods of time.

Assessment of nutritional status is usually directed primarily at the high-risk age group of children <5 years of age. Monitoring of this vulnerable group provides sentinel information on the community at large. As discussed above, weight-for-height is the established index for detecting acute undernutrition. Standard methods of estimating the nutritional status of populations have been developed (42,59). Using the cluster-sample method, data are collected on the height, weight, and presence of edema for children 1-5 years of age in each of 30 clusters selected from the population. Screening for suspected vitamin deficiencies can also be included in this survey. If survey data indicate a need for supplemental and/or therapeutic feeding, screening of the entire high-risk age group is then required for enrollment in the program.

ONGOING SURVEILLANCE

Ongoing surveillance provides two types of data: **trend** data on mortality and nutritional status over time and **morbidity** data on conditions of public health importance.

Mortality and nutritional status can be monitored as described under short-term assessment for camps and settlements. The estimation of population-based mortality may require demographic surveys which rely on recall of deaths by family members. The value of monitoring the effectiveness of programs can be seen from the graph in Figure 2, which shows mortality and undernutrition prevalence over 8 months in the Tigrean and Kampuchean refugee populations discussed earlier (29). Although these data were plotted several years later, had the trends been closely observed at the time of these relief operations, appropriate corrective action might have been taken in the case of the Tigrean population (which experienced prolonged undernutrition prevalence and high mortality).

Data on morbidity are available from four sources—inpatient admissions, outpatient consultations, outbreak investigations, and population-based surveys. In famines involving large populations, collection of quality data from selected, or sentinel, inpatient and outpatient facilities has proven useful in identifying disease problems in the community.

Data collected on morbidity should be limited to information about diseases for which control programs are under way or for conditions that, if found, would lead to specific interventions. Listed below are some of the important conditions that should be considered when a surveillance system for famine areas is being developed.

Major causes of mortality

Undernutrition
Measles
Diarrhea (including cholera)
Dysentery
Pneumonia
Malaria
Meningitis

Epidemic diseases that require immediate action

Measles
Meningococcal meningitis
Cholera
Malaria
Polio
Typhus

Specific nutritional deficiencies

Xerophthalmia
Scurvy
Beriberi
Pellagra
Microcytic anemia (iron deficiency)
Macrocytic anemia (folate and B12)

Note that almost all these conditions are preventable if the programs discussed earlier are implemented.

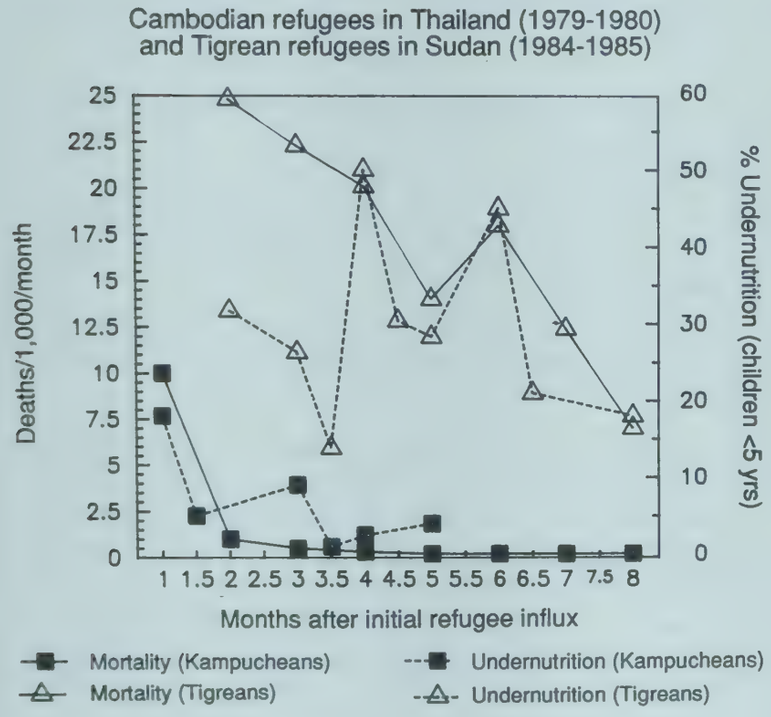
In the establishment of a disease surveillance system for famine areas, the five principles below are useful.

- Data collection should be limited to information required for monitoring health status (mortality and nutritional status) and key medical conditions for which interventions are available and feasible and for which diagnoses can reliably be established. For example cough and fever is preferable to pneumococcal pneumonia.
- The reporting form should be limited to one page if possible and should be formatted for data entry and compilation.
- Reporting frequency should depend on need—daily (in acute emergencies), weekly, or monthly. In stable,

post-famine situations, monthly reporting is usually adequate.

- Reporting compliance should be monitored, with active follow-up for missing reports. Negative reporting—that is, the filing of reports even if there are no cases—is essential.
- Analysis should be rapid, with immediate actions taken in response to identified problems. Feedback of analyzed data to implementers, data providers, and program decision-makers should be prompt. In Somalia, this feedback has been successfully carried out via a monthly newsletter to staff in refugee camps.

FIGURE 2. Crude mortality rates and prevalences of childhood undernutrition



Prevention of Famines

As discussed earlier, early-warning systems offer considerable hope in accelerating the response to a prefamine situation. Experience has shown, however, that the knowledge afforded by data generated by an early-warning system do not necessarily stimulate potential donors to act. For famine-prone countries, there is much that can be done in advance of a possible famine, including storage of food reserves, plans for mobilization of transport, and training of civil servants in disaster relief, but these measures are difficult to enact in the countries that need them most.

The prevention of famine requires the combined efforts of agriculturalists, economists, nutritionists, and planners. Since famines generally strike the poorest of the poor in developing societies, measures that are required include changes in land ownership and tenure, an increase in government resources devoted to rural development, and the encouragement of agricultural production, as well as taxation and price policies that bring about better equity (60,61).

Relief efforts need reliable information in place on the availability of local foods, local food tolerances and preferences, transportation available for the distribution of food relief, quality of the civil infrastructure of the latter, and the nature and extent of the food shortage. The lack of such information has been responsible for much confusion and inefficiency in the past (8).

Need for Further Study

The need for study of the causes, prediction, control, and prevention of famine is too vast to cover here, spanning as it does so many disciplines. In the field of famine epidemiology, further field studies are needed in the following areas:

1. Improvement in nutrition and mortality surveillance systems;
2. Prospective studies of the correlation between undernutrition and mortality;
3. Studies of the association between certain micronutrient deficiencies and morbidity and mortality;
4. Prospective studies of the daily energy and protein needs of certain at-risk groups within populations;
5. Trials of certain supplementary and therapeutic feeding protocols in the prevention and treatment of protein-energy malnutrition.

Conclusions

Famine is largely a preventable phenomenon. The failure to provide sufficient food for consumption by all members of a population is the result of a number of interacting factors—some unavoidable natural calamities, but most caused by humans. Famines will continue to occur given the continued political turmoil, economic decline, and rapid population increases in some parts of the world. The following assessment, made in 1972, remains true today (62):

"Famine occurs months or even years after the primary event. Famine relief, then, is an admission of failure to tackle the primary event. Famine is the disaster situation with a long enough incubation period that it should never occur on a mass scale."

If and when famines do occur, their impact will be minimized if relief programs include the following components:

1. Effective nutritional and mortality surveillance;
2. Community-based provision to all families of adequate general rations;
3. Appropriate preventive actions, such as measles vaccination and control of diarrheal disease;
4. Supplementary and therapeutic feeding programs, when indicated; and
5. Immediate interventions to rehabilitate the food production and distribution systems.

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Air Pollution

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Background and Nature of Acute Episodes of Air Pollution

Sources of Air Pollutants

Pollutants may enter the air from natural or synthetic sources. Air has always carried such natural pollutants as pollens, spores, molds, yeast, fungi, and bacteria. Forest fires, windstorms, volcanic eruptions, and droughts all cause smoke, dust, and other pollutants to enter the air. Yet all the pollution wrought by nature counts for little when compared with the effects of pollutants associated with human activity (1). The major sources of synthetic air pollutants include the burning of fossil fuels, particularly coal—in operations such as power plants—in combination with emissions from smelters, steel, and other heavy manufacturing. Emissions from mobile sources such as automobiles, trucks, and airplanes constitute another major source of air pollutants. The primary pollutants from these major sources are sulfur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO), suspended particulates, ozone, hydrocarbons, acid aerosols of sulfates and nitrates, and heavy metals. Although emissions from chemical manufacturing may affect the area immediately surrounding the source, they do not have the same impact on regional air pollution as the aforementioned pollutants.

Factors Contributing to a Buildup of Concentrations of Air Pollution

Adverse meteorologic conditions cause air pollutants to accumulate. Low wind speeds (< 4 km/hour [7 mph]) provide inadequate movement for the dispersion of air pollutants, which creates a condition referred to as stagnation.

A further complication is provided when a "temperature inversion" occurs at or not far above the earth's surface. This is a reversal of the atmosphere in which temperature in a layer rises with height instead of decreasing. The most common cause is nighttime cooling of a stratum near the ground when there are light winds with little mixing and clear skies to permit the free radiation of surface heat. However, an inversion can also result or be intensified because of complicated large-scale meteorologic events usually associated with high atmospheric pressure. Within an inversion layer, vertical atmospheric motions that normally might disperse pollutants are minimized, and sometimes an inversion layer hovering over a city acts as a lid. This keeps pollutants from escaping upward, and upward is the only direction for them to go when wind speed is insufficient to disperse them horizontally (2).

Areas with a high frequency of both low wind speeds and inversions have the greatest potential for a buildup of air pollution concentrations. The problem is intensified in areas in which air movement is restricted by surrounding hills or mountains.

Historical Review of Acute Episodes of Air Pollution

Humans have been exposed to synthetic sources of air pollution since they lit the first fire. The air of many of the earliest towns was rife with smoke and noxious odors emanating from trades such as tanning. With the general use of coal, air pollution began to be a major problem. Wood was the prime source of heat throughout early Europe. By the 1200s, however, forests near settlements were depleted, and a new fuel was needed. Increasingly, Europe followed the example of Asia, whose coal-burning technology was described by such travelers as Marco Polo.

The use of coal fouled the air so badly that in 1273 England's King Edward I passed a law prohibiting the use of at least one type of coal, and in the early 1400s Henry V formed a commission to oversee the use of coal in the city of London. In 1661 Charles II ordered the scientist John Evelyn to survey the effects of the increasing air pollution over the city. Evelyn recognized the relationship between the "dismal cloud" over London and a number of fatal diseases, but his warnings of the need for controls were ignored.

Buildups of air pollution had sporadically afflicted towns after the introduction of coal, but urban centers still had fairly small populations, industry operated on a small scale, and the outpourings of industrial contaminants were not yet the norm. Early episodes of air pollution were thus relatively minor in effect. By the late 1800s industry was booming,

larger and larger populations were concentrating in cities, and increasing amounts of chemical pollution were entering the air. As a result, in December 1873, when weather conditions fell into a certain sequence, a thick cloud of pollutants gathered over London. This episode resulted in 1,150 deaths, making it one of the earliest air pollution disasters (3).

Since 1873 the industrialized world has known at least 40 episodes in which sudden buildups of air pollution have caused widespread casualties. London experienced similar episodes in January 1880, February 1882, December 1891, and December 1892. During the autumn of 1909, Glasgow, Scotland, suffered an acute episode, resulting in 1,063 deaths. In 1911 in preparing a report on the Glasgow episode, Dr. Harold Antoine Des Vouex coined the word "smog" as a contraction of smoke-fog (4).

In 1911 the highly industrialized valley of the Meuse River in Belgium experienced a thermal inversion that trapped all pollutants along the 15-mile length of the valley. The resultant buildup of pollution caused undetermined scores of casualties. Virtually the same weather conditions recurred in December 1930. Few air pollution controls had been established in the interim, and by 1930 the valley had become even more industrialized. The latter episode caused 63 deaths, and 6,000 persons became ill (5).

In October 1948, the first reported air pollution disaster in the United States occurred in Donora, Pennsylvania. Donora is situated in a horseshoe-shaped valley of the Monongahela River, and the city contained large plants that produced steel, wire, zinc, and sulfuric acid. A fog closed in on the area, accompanied by a pollutant-trapping thermal inversion. Twenty deaths were attributed to the resultant buildup of air pollutants, and 5,190 persons became sick (6).

In December 1952 London experienced another air pollution episode, which resulted in an estimated 4,000-8,000 deaths (7).

Although other acute air pollution episodes have occurred since 1952 in such places as New York City; Los Angeles, California; Birmingham, Alabama; and Pittsburgh, Pennsylvania, none were of the magnitude of the episodes mentioned above. These latter episodes resulted in few deaths and relatively mild symptoms in a small subset of the population (8-10).

Since the United States Congress passed the Clean Air Act in 1963 and the Clean Air Act Amendment in 1970, the air quality in the United States has greatly improved, and the number of acute episodes of air pollution has decreased substantially (11).

Factors That Influence Morbidity and Mortality

Several studies conducted after acute episodes of air pollution provide some information on the type of adverse effects experienced during the episodes, the population segments at highest risk for having adverse effects, and the nature of the air pollutants thought to be responsible for effects observed. Although these studies have varied in depth and design, findings have been consistent.

Meuse Valley, Belgium, 1930

The results of an investigation launched shortly after the Meuse Valley episode in 1930 indicated that elderly persons not necessarily in poor health accounted for most of the 63 deaths (although no age-specific rates were determined). Also among the dead were relatively young persons who had preexisting disease of the heart and lungs. Persons from these two groups were among the first to die. However, as the episode continued, thousands of otherwise healthy people became ill, and some of them died. No single pollutant could be blamed, and no pollutant ever reached the dose determined to be lethal in laboratory settings. Rather, it was thought that pollutants acting in combination intensified one another's effects. Probably the worst offender was the combination of sulfur dioxide and sulfuric acid mist. The investigators also judged that the nitrogen oxides from fuel combustion and industrial processes contributed to the casualties. It was further thought that particles of metallic oxides made the polluted air more hazardous (12).

Donora, Pennsylvania, 1948

The most thorough investigation of an episode of air pollution was carried out by the U.S. Public Health Service and the Pennsylvania Department of Health after the 1948 episode in Donora, Pennsylvania. From these studies it was determined that 5,190 Donorans—almost 43% of the people in the area—were made ill to some degree by the smog.

The principal health effect was essentially an acute irritation of the respiratory tract and, to a lesser extent, of the digestive tract and eyes. During the smog, 33% of all the people in Donora developed coughs, the most common single symptom. Other reported symptoms, in descending order, were sore throat, constriction of the chest, shortness of breath and difficulty in breathing, headache, nausea and vomiting, smarting of the eyes, tears, and nasal discharge. Relatively few of the people affected by the smog escaped with an ailment considered mild. Of every 100 persons who became ill, at least 24 suffered severe symptoms and 39 experienced symptoms of moderate severity (as determined by how disabling the symptoms were, how much medical care was required, and how long symptoms lingered after the smog).

The morbidity rate rose with increasing age; 31% of persons age 20-24 years, 55% of persons 40-44 years of age, and 63% of persons 60-65 years of age became ill. The severity of illness also rose with increasing age.

Preexisting disease of the heart or lungs was a major factor in the 20 deaths from the smog, but not all of the deceased had histories of chronic disease. In four instances the decedents had been in excellent health until they were affected by the smog. Autopsy reports for two of the victims listed bronchitis, pulmonary edema, and hemorrhage as the cause of one death and cor pulmonale as the cause of the other. The decedents ranged in age from 52 to 84 years (13).

A decade after the disaster, investigators reassessed the impact of the acute episode on the health status of the population of Donora. Persons who had become ill during the smog subsequently died younger and were ill more often than those who had not been affected during the episode. For those who had suffered severe complaints during the smog, the death rates were particularly high. Even persons

who had no history of heart disease but who developed symptoms during the smog continued to suffer impaired health (14).

The investigators reported that a combination of pollutants had probably caused the disaster. Sulfur oxides in combination with airborne particles of metals and metallic compounds were thought to be the likely cause of the injuries incurred during the episode of smog (13).

London, England, 1952

Although no in-depth study was done immediately after the London smog episode, an analysis of death certificates and reports from several physicians who attended the ill during this episode revealed findings similar to those reported in the study in Donora, Pennsylvania.

The first evidence of illness and death associated with the smog was among livestock. The opening of the Smithfield Club Show, one of Britain's most important livestock exhibitions, coincided with the onset of the air pollution episode. The livestock were prize cattle brought from the best herds in the United Kingdom, and they were young, fat, and in prime condition. Despite the animals' good health, some 160 became ill soon after the onset of the smog. At first the breathing of a cow or bull increased slightly, causing the animal to keep its mouth open. As the smog worsened, breathing became faster and more labored. Many beasts in marked distress hung out their tongues, panting harder and harder like dogs. The animals grew feverish and refused to eat. Sixty developed severe symptoms and required major veterinary treatment. A dozen of the cattle—in pain and beyond hope—were slaughtered, and another animal died. Detailed examination of the carcasses indicated that the animals had suffered extreme respiratory irritation, resulting in emphysema, pneumonia, and pulmonary edema.

The most common symptoms among humans were respiratory disorders, chiefly an extremely irritating cough sometimes accompanied by gray, pus-thickened phlegm. Patients were debilitated by shortness of breath, convulsive coughing, and painful gasping of air. Many developed cyanosis and wheezing. The most severely ill patients were those with histories of heart or respiratory disease. At the Emergency Bed Service, requests on behalf of patients with heart problems were more than three times normal and for patients with respiratory disorders, nearly four times normal. For patients with both heart and respiratory problems, demand was even greater, and these people were the first to die. Typically, those ill from the smog took from 4 to 9 days to recover.

During the smog period 2,851 persons died in excess of the norm. In the following week an excess of 1,224 other deaths were attributed almost exclusively to the smog. A study of death certificates for the months after the smog showed evidence of excess deaths occurring up to 12 weeks after the episode, bringing the estimated total deaths attributed to the episode to 8,000. Persons $\geq$ 65 years of age had a death rate 2.7 times above normal, but those age 45-64 had even higher rates, 2.8 times the normal rate for that age group. Infants $<$ 1 year of age had rates 2.0 times normal. No human autopsy reports were provided among the reported data

The coroner's statistics showed that during the episode, deaths occurred both inside and outside the home. Twenty-eight heart patients died suddenly while at their place of work or elsewhere outside their homes. Some 105 persons suffered heart attacks while engaged in sedentary activities inside their homes.

The harmful agents in the London episode were thought to be a combination of irritant pollutants—principally smoke and sulfur dioxide. Smoke concentrations during the smog were measured at 5 times London's normal level. Particles of soot showed up universally in the sputum. The general level of sulfur dioxide throughout the smog was about 6 times normal; in some areas, the sulfur dioxide concentration rose to 12 times the norm. An estimated 60% of the total pollution in the smog arose from domestic fires in which soft coal was burned. The remainder of the pollution came from commercial sources and motor vehicles (7,14). Stronger air-pollution-control measures were instituted after the acute episode, with particular attention given to the control of smoke and suspended particulates.

New York City, 1953

A statistical study of emergency room visits and death certificates after an acute episode of air pollution in New York City in 1953 established that during the smog a substantial number of persons had suffered cardiac and respiratory distress. Moreover, results of the final study indicated that 175-260 persons died because of the smog (8).

Information about these various episodes shows that the population at greatest risk of morbidity and mortality is the group with preexisting respiratory and cardiac conditions. These people are the first to develop symptoms and among the first to die. As the episode continues, other segments of the population—particularly the old and the very young—become symptomatic, and many die. The primary symptom is respiratory irritation manifested by cough, but gastrointestinal symptoms such as nausea and vomiting may also occur. Cardiopulmonary complications are the major cause of death, and the limited information available from autopsy reports suggests that infection, pulmonary edema, and hemorrhage may contribute to death. The only study giving location and activity of the decedents before they died was a report on the London episode of 1952, and that information showed no protective effect from remaining indoors. However, since a major source of pollution in that episode was domestic coal fires, the indoor environment may also have been heavily polluted.

The follow-up study in Donora, Pennsylvania, suggests that persons who experience symptoms during an acute episode of air pollution may be at higher risk than other persons for subsequent illness and early death. The more severe the symptoms during the episode, the more severe the residual effect.

Although the technology for measuring pollutants at the time of these episodes was quite crude as judged by today's standards, there was some consistency in the assessment that a combination of pollutants rather than a very high level of a single pollutant was probably responsible and that the principal hazard was sulfur dioxide in combination with metallic particles.

Public Health Implications

Prevention and Control Measures

The principal steps to follow to prevent injury and death associated with acute air pollution episodes are as follows.

- To recognize meteorologic conditions such as low wind speed and thermal inversions that might lead to a buildup of air pollutants.

  Meteorologists with the National Oceanic and Atmospheric Administration (NOAA) have an ongoing monitoring program for recognizing the presence of such adverse conditions in the United States. The NOAA meteorologists alert the United States Environmental Protection Agency (EPA) when such conditions are identified.

- To measure air pollution levels during adverse meteorological conditions and prevent the buildup of hazardous levels of pollutants.

  EPA, in conjunction with state and local air pollution agencies, has an ongoing air monitoring network that covers many large industrial regions of the United States. Thus, a system is in place to monitor the ambient air in these areas during adverse meteorologic conditions. If an inversion or air stagnation occurs outside the monitoring network, air monitoring resources from Federal, state, or local agencies may be used.

  When air pollution levels exceed the short-term standard, the EPA or the responsible state agency issues an air pollution alert. EPA has the authority to shut down industry if it judges that continuing emissions from such sources during the adverse meteorologic conditions would pose a hazardous air pollution situation (15).

- To alert susceptible segments of the population to take appropriate action and minimize their exposure to hazardous levels of pollution.

  The EPA, as well as some state health departments, has developed guidelines to be issued during air pollution alerts. These guidelines contain precautions that susceptible members of the population should take under various conditions of air pollution buildup (15). The Pollution Standard Index developed by EPA is shown in Table 1.

Information Dissemination

Often an air pollution episode goes unrecognized by a major portion of the population in the area involved. During the episodes in London and New York, many of the illnesses and deaths were not reported as being associated with the poor air quality.

The media, particularly television and radio, can play a vital role in informing the public of the air pollution episode and the precautions that should be taken. It is important to have a communication network whereby persons identifying adverse meteorologic conditions and a buildup of air pollution can convey this information through appropriate channels to the media for broad dissemination.

Surveillance

The EPA conducts an ongoing surveillance program of ambient air quality throughout the United States through its air monitoring network. The resulting data permit potential problem areas to be identified in the event of adverse meteorologic conditions. Air quality in the United States has improved considerably since the passage and enforcement of the Clean Air Act, and the number of air pollution alerts has decreased dramatically (11).

Research Recommendations

Although our understanding of risk factors associated with acute episodes of air pollution episodes has been greatly augmented through studies, the following research questions should be addressed in the event of another major episode.

- Ongoing air pollution measurements of a broad spectrum of agents should be taken during an episode to determine the specific pollutants that might be associated with death and injury. In previous episodes, often no measurements were taken during the event. In the few situations in which measurements were taken, they were limited to a narrow class of pollutants, and the measurement technology used was—by today's standards—antiquated and imprecise.

- A reporting system should be established during an episode to assess more accurately the number of illnesses and deaths associated with the episode, as well as the nature and severity of the illnesses. This reporting network should involve private physicians, hospital inpatient and outpatient admissions, a sampling of households, and reports from medical examiners and coroners.

- Case-control studies should be conducted to determine whether limiting exercise and remaining indoors provide protection from illness or death. Such studies could also confirm or negate the risk factors associated with morbidity and mortality in previous episodes.

- A cohort of affected individuals should be followed over time to determine whether they are at higher risk of having chronic effects than unaffected members of the exposed population.

Summary

Acute episodes of air pollution are caused by a buildup of pollutants during periods of low wind speed (< 4 km/hour) or temperature inversions. Increased industrial emissions—coupled with increased aggregation of people into urban areas during the latter part of the 19th century—created circumstances whereby large numbers of people were exposed to hazardous air pollution during adverse meteorologic conditions. Since 1870, at least 40 episodes of air pollution have been accompanied by death and illness. The largest number of deaths reported from an episode occurred in London, England, in 1952, when an estimated 8,000 deaths were attributed to the episode.

Persons at greatest risk of morbidity and mortality from such episodes are those with preexisting respiratory and car-

TABLE 1. Comparison of PSI* values with pollutant concentrations, descriptor words, generalized health effects, and cautionary statements

Index value	Air quality level	POLLUTANT LEVELS: TSB (24-hour) pg/m³	S_{02} (24-hour) pg/m³	CO (8-hour) mg/m³	03 (1-hour) pg/m³	N_{02} (1-hour) pg/m³	Health effect description	General health effects	Cautionary statements
500	Significant harm	1,000	2,620	57.5	1,200	3,750		Premature death of ill and elderly. Healthy people will experience adverse symptoms that affect their normal activity.	All persons should remain indoors, keeping windows and doors closed. All persons should minimize physical exertion and avoid traffic.
400	Emergency	875	2,100	46.0	1,000	3,000	Hazardous	Premature onset of certain diseases in addition to significant aggravation of symptoms and decreased exercise tolerance in healthy persons.	Elderly and persons with existing diseases should stay indoors and avoid physical exertion. General population should avoid outdoor activity.
300	Warning	625	1,600	34.0	800	2,260	Very unhealthful	Significant aggravation of symptoms and decreased exercise tolerance in persons with heart or lung disease, with widespread symptoms in the healthy population.	Elderly persons with existing heart or lung disease should stay indoors and reduce physical activity.
200	Alert	375	800	17.0	400¶	1,130	Unhealthful	Mild aggravation of symptoms in susceptible persons, with irritation symptoms in the healthy population.	Persons with existing heart or respiratory ailments should reduce physical exertion and outdoor activity.
100	NAAQS	260	365	10.0	160	†			
50	50% of NAAQS	75§	80§	5.0	80	†	Moderate		
0		0	0	0		†	Good		

* Pollution Standard Index.
† No index values reported at concentration levels below those specified by "Alert Level" criteria.
§ Annual primary NAAQS.
¶ 400 pg/m³ was used instead of the 0_3 Alert Level of 200 pg/m³ (see text).

diac conditions. They are among the first to have symptoms and among the first to die. The old and the very young are also at increased risk of becoming ill and dying. The primary effect is respiratory irritation manifested by cough and chest discomfort, but gastrointestinal symptoms such as nausea and vomiting may also occur. Cardiopulmonary complications are the major cause of death, and information available from a limited number of autopsy reports suggests that pulmonary edema, infection, and hemorrhage may contribute to death.

Findings from one study suggest that persons who experience symptoms during the episode may be at higher risk than others for subsequent illness and premature death. The more severe the symptoms during the episode, the more severe the residual effect.

Although the technology for measuring pollutants at the time of these earlier episodes was quite crude by today's standards, some consistency was shown in the assessment that a combination of pollutants rather than a very high level of a single pollutant was responsible, and that the principal hazard was sulfur dioxide in combination with metallic particles.

Death and injury from episodes of air pollution can be prevented by identifying low wind speed and temperature inversions, monitoring air pollutants during the period of adverse meteorologic conditions, limiting pollution emissions when indicated, and advising susceptible segments of the population on preventive measures to take under varying air pollution conditions. The passage of the Clean Air Act and its amendments has led to improved air quality in the United States, and the number of episodes of air pollution has decreased substantially.

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Industrial Disasters

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Introduction

Historically, with the exception of military actions, natural causes have accounted for most disasters. However, growing industrialization has led to a new type of disaster—the industrial disaster—as a result of storage, processing, and transportation of large amounts of highly flammable, explosive, and toxic chemicals. In this chapter we discuss fires or explosions in industrial settings or in transit and acute chemical or radioactive releases from point sources. We restrict this discussion to events with immediate public health impacts, and exclude the contamination of food by chemical or radioactive substances.

Factors That May Contribute to Industrial Disasters

Several factors contribute to the occurrence of industrial disasters and their impact on public health. These factors relate both to the chemicals involved and the population exposed. If industrial disasters are to occur, reactive or toxic chemicals must be in situations in which they can be released, explode, or catch fire. Natural phenomena, such as earthquakes, may initiate releases, but usually operator error, equipment failure, or facility-related factors trigger the event. It is estimated that over 62% of hazardous material spills during transport are related to human error (1).

If a release is to have a public health impact, individuals must be exposed to the chemicals, fire, or products of a blast. Generally, workers at the scene of the incident are at initial and greatest risk. First responders (e.g., firefighters) can also be endangered if they are improperly protected. An often-forgotten occupational group at risk are health-care workers, who may be exposed far from the site of the release if contaminated persons are not decontaminated before they are brought to a medical facility.

Unless a large release occurs, people who live near the release area are less often severely physically affected than workers. However, hundreds of evacuations and orders to stay indoors occur annually in the United States as a result of actual or threatened releases (2), and these can result in psychological stress. The magnitude of the release, distance of residents from the incident, actions taken by residents, weather conditions, and type of housing are often critical factors in determining the effects on community residents.

Review of Industrial Disasters

The few recorded industrial disasters that have caused large numbers of deaths have occurred throughout the history of industrial development. Table 1 lists some of the major industrial disasters of this century (3). Most of these, such as the San Juan Ixtaheupec disaster, have involved fires or explosions. Others, such as the methyl isocyanate release in Bhopal, India, illustrate that the release of toxic chemicals—even without explosion or fire—can also have devastating consequences. A description of the release in Bhopal, India, follows.

Bhopal, India

The release at Bhopal, India, provides an excellent example of factors that contribute to an industrial disaster (4). The Union Carbide of India, Limited, plant in Bhopal manufactured carbamate pesticides using a process that required large amounts of methyl isocyanate (MIC), an extremely irritating chemical that can cause severe acute respiratory problems. Over the 15 years the plant was in operation, the city of Bhopal had become densely populated and expanded up to the boundaries of the plant. On December 3, 1984, over 30 tons of MIC were released into the air. The cause of this release has not yet been firmly established; most likely, a substantial quantity of water entered a large MIC storage tank, starting an exothermic reaction. Several safety systems designed to prevent a major release were inoperative, under maintenance, or not activated by workers. As the chemical reaction progressed, a pressure-release valve burst, and the contents of the tank were released.

Starting at about 12:30 a.m. and continuing for about 2 hours, a cloud of MIC slowly spread over Bhopal. A light wind blowing toward the densely populated parts of the city and an atmospheric inversion kept the toxic cloud close to

the ground, increasing the number of people exposed. Warning systems for the community were not activated until hours after the release began, and many individuals died in their sleep. The residents of Bhopal knew little about the chemicals being manufactured in the plant, and many ran toward the plant as they became symptomatic. The streets were poorly lit and crowded, making evacuation of the area difficult.

Over the next several days, the most common complaints were irritated eyes, choking sensations, breathlessness, and weakness (5). Occupants of a densely populated area of small shacks directly across the street from the plant site were most severely affected (accounting for most of the deaths), but residents in areas further away also became ill. Initial estimates indicated that >2,000 of the 200,000 residents exposed to the MIC died, and 50,000-200,000 were affected severely enough to seek medical care. In 1988, authorities reported that nearly 3,000 people had died, and an additional 1,700 were expected to die by 1995 as a result of the release (6).

The Indian government has coordinated medical follow-up, which has included establishing a clinic near the most severely affected areas and performing a door-to-door survey to interview surviving residents and to refer them to the clinic for evaluation. Attention has focused on possible pulmonary disease and adverse reproductive outcomes due to MIC exposure.

This release illustrates many important points about industrial disasters. It was not caused by a single factor; a number of contributing events had to occur for the MIC to be released and have such a great public health impact. Human error and lack of adherence to well-defined safety rules played a substantial role. Much of the morbidity and mortality could have been prevented had a less toxic substance been released, had the quantity released been less, had the population near the site been smaller, had the housing been of better quality, had the wind blown in a different direction, had appropriate warnings been given, or had the population been better educated.

Data on Acute Chemical Releases, United States

The Bhopal release, unfortunately, is not an isolated incident. Although complete data on the numbers and outcomes of acute chemical releases are unavailable, there are some limited surveillance systems for such events. The Acute Hazardous Events Data Base (AHE), a partial list of releases involving toxic chemicals in the United States, was compiled by the Environmental Protection Agency and its contractors (7). In the period 1980-1985, the database recorded 6,928 separate incidents, of which 75% occurred in a plant and 25% took place while materials were in transit; 468 of these events resulted in deaths or injuries. In 1986, events reported to the National Response Center, the Department of Transportation's Hazardous Materials Information System, and AHE, indicated that 587 releases (about 1.6 incidents/day) had occurred that led to deaths, injuries, or evacuations (2). Fifty-

TABLE 1. Major industrial disasters

Date	Place	Event	Result
9/21/21	Oppau, Germany	Explosion at a nitrate manufacturing plant destroyed plant and nearby village	561 deaths; > 1,500 persons injured
4/16/47	Texas City, Texas	Explosion in freighter being loaded with ammonium nitrate	561 deaths; much of city destroyed
7/28/48	Ludwigshafen, Federal Democratic Republic of Germany	Vapor explosion from dimethyl ether	209 deaths
7/10/76	Seveso, Italy	Chemical reactor explosion released 2,3,7,8-TCDD	100,000 animals killed; 760 people evacuated; 4,450 acres contaminated
2/25/84	Cubatao, Sao Paulo, Brazil	Gasoline leak from a pipeline exploded and burned nearby shanty town	> 500 deaths
11/19/84	San Juan Ixtaheupec, Mexico City, Mexico	5 million liters of liquefied butane exploded at a storage facility	> 400 deaths; 7,231 persons injured; 700,000 evacuated
12/3/84	Bhopal, India	Release of methyl isocyanate from pesticide plant	> 2,000 deaths; 100,000 persons injured

Source: (Reference 3.)

six percent of these events were related to the transport of chemicals. Most of these events were individually associated with only a few deaths or injuries; however, had circumstances differed slightly, many of these incidents could have had far more serious acute public health effects. Moreover, illnesses with long latency periods—such as cancer—are not quantified in these data.

Factors That Influence Morbidity and Mortality

If a hazardous substance is released during an industrial emergency, the toxicity of the substance and the routes and amounts of human exposure are important determinants of the potential public health effects. Planning, preparation, and appropriate emergency response are the keys to minimizing human exposures to toxic chemicals.

Human Exposure and Health Impact

The health effects from a toxic chemical may differ depending on the route of exposure. Inhalation is often the most obvious exposure route, particularly during the acute phase of a release. However, with the passage of time, dermal exposure from touching contaminated objects or gastrointestinal exposure from ingesting contaminated food and water may become more significant routes of exposure than inhalation.

Dose is also an important determinant of health impact. Many chemicals, particularly noncarcinogens, are believed not to cause adverse effects if exposure is below a certain "threshold." Different target organs often have different thresholds. Whether or not a threshold exists, many effects of chemicals show a relationship between dose and response. It must also be recognized that industrial incidents may cause serious injury or death not only through toxic effects of released chemicals but also from burns or injuries from fires and explosions.

Prevention Measures

Whereas flammable and explosive materials have been recognized as serious hazards for some time and some control measures have been taken, less attention has been paid to prevention of simple releases of chemicals. The storage and handling of industrial material have been inadequately scrutinized, and existing regulations—including transportation regulations—are not well enforced. Strengthening and enforcing these laws is essential for prevention of unintentional chemical releases.

Although ideally, acute chemical releases should be prevented altogether, measures should also be taken to decrease human exposure when releases do occur. As has already been stated, the first people at risk from an incident are usually the workers in the facility. Various means can be used to protect them, including engineering controls on industrial processes, monitoring systems to detect and control releases, and personal protective equipment (e.g., respirators). In addition, proper training is fundamental to any prevention program. This training must include information about the substances used in the industrial facility, the potential hazards associated with these substances, and the appropriate means for controlling and preventing inadvertent releases.

Morbidity and mortality for persons who respond to emergency situations (e.g., firefighters) can be decreased if precautions are taken with protective body coverings, respiratory protection, and decontamination procedures. Such persons also need to be educated about such matters as the chemicals they are likely to encounter, appropriate response techniques, and decontamination procedures.

The nearby population must also be protected. Planners should consider the location of the plant in the context of the surrounding communities. Local zoning ordinances govern the location of facilities and residential areas; however, uniform regulations or recommendations on the siting of potentially hazardous facilities have generally not been published. Even if facilities are sited in "safe" locations, a safe location can become less so over time, as the uses of a facility change and the populations near it grow. Transportation routing for hazardous materials must consider not only the populations that would be exposed in the event of an incident, but also the road conditions, availability of emergency-response teams, and existence of alternative routes.

EMERGENCY PREPAREDNESS

Emergency preparedness complements prevention strategies. This preparedness has two phases: information collection and contingency planning. Information that should be readily available to workers and emergency-response personnel includes not only the types and quantities of chemicals on the plant site, but also typical symptoms of exposure and appropriate medical treatments.

The 1986 Superfund Amendments and Reauthorization Act (SARA) requires that facilities with certain potentially hazardous chemicals identify these chemicals (and often provide information about them) to local emergency planners and other officials. SARA also requires that emergency planning committees—including, e.g., elected officials, police and fire personnel, and public health professionals—be established at the local level. These committees are responsible for developing emergency-response plans. By law, such plans must include identification of facilities and transportation routes with extremely hazardous substances, on- and off-site emergency-response procedures, emergency notification procedures, methods for determining that a release has occurred and the area and population that will probably be affected, description of community and industry emergency equipment and facilities and the identity of persons responsible for them, evacuation plans, description and schedules of training programs for emergency-response personnel, and methods and schedules for rehearsing emergency-response plans. These provisions of SARA, if successfully implemented, will be an important step toward using emergency preparedness to decrease public health consequences of chemical releases (8).

EMERGENCY RESPONSE

Although it is imperative that attention be given to primary prevention of explosions, releases, and fires, realistically, some efforts must also be directed at emergency responses. Such responses must be organized and directed in a manner that will minimize deaths, illnesses, injuries,

and environmental damage resulting from releases of hazardous material.

Initial efforts in an industrial accident should be aimed at protecting the people on site, those responding to the incident, and the potentially affected residents of the community. For persons in the community, this may mean evacuation or orders to stay indoors with windows closed until better control is established.

Early efforts should also be directed toward identifying the types, quantities, and concentrations of the chemicals released. Sometimes this information is available from a transportation manifest or from plant managers. Unfortunately, proper identification sometimes requires outside consultation or time-consuming chemical analysis of the material. If possible, air and (if appropriate) water samples should be gathered as the situation evolves. When the released chemicals have been identified, appropriate protective gear for responders and medical treatment and follow-up for exposed persons can be determined. After the chemicals have been identified, chemical-specific control measures should be instituted.

Although emergency response to chemical incidents may seem simple and direct, actual responses are often more complicated. The need to make quick decisions and to respond rapidly—even though adequate information is not available, there are difficulties with information dissemination, the division of responsibilities between responding agencies is unclear, no overall leader has been designated, and agencies or individuals have divergent priorities—is anything but simple. Major efforts must be directed at proper preparation, training, and coordination.

Epidemiologic Investigations

Epidemiologic investigations of industrial disasters can include the following: surveillance and description of the public health impact of the disaster, examination of the effects of emergency response actions on public health impacts, evaluation of relief efforts, analysis of risk factors for adverse health effects, clinical investigations of effects of diagnostic and treatment approaches, description of the natural history of the acute health effects, population-based studies of long-term health effects, and assessments of the psychosocial impact of the disaster. The results of epidemiologic studies can help health-care practitioners by providing information useful for individual patient care, helping match resources to needs, and contributing to primary prevention of adverse outcomes and to contingency planning. These studies also help emergency-response planners and coordinators by indicating which strategies are effective in reducing the public health impact of disasters and ways of improving public compliance with directives.

The nature and extent of a disaster may affect the ability to conduct an epidemiologic investigation. Most epidemiologic studies of the affected population require that the potentially affected individuals be defined. Displacement and disruption caused by industrial disasters may make this difficult. For example, in Bhopal, no accurate census of the city existed. Normal record systems were disrupted; burial certificates were not issued for many of the dead, and the city's primary teaching hospital kept no medical records during the first day of the incident. An accurate assessment of the exposed population may require a combination of sources of data, e.g., door-to-door census-taking in the affected areas, as well as reviews of medical, death, and tax records.

Many post-disaster epidemiologic studies require an assessment of exposure. For some toxic substances, each individual's exposure can be determined by biological monitoring (9). For example, shortly after the 1986 uranium hexafluoride release at Gore, Oklahoma, investigators monitored urinary uranium levels to estimate the acute exposure. These estimates of exposure could then be used in predicting possible longer-term health consequences for the exposed individuals. Unfortunately, practical biological monitoring methods do not always exist for the relevant chemicals, and epidemiologists must rely on modelling of exposure. In the absence of biological data, exposure can best be modelled when immediate environmental sampling provides information on the chemical concentrations present in the affected area at the time of the disaster. Epidemiologists should coordinate with the people responsible for assessing the environmental contamination from the disaster. This is important to ensure that information is collected in a way that will be useful for assessing human exposure at different distances from the site of the release.

When appropriate environmental data are available, estimation of the amount of air breathed, exposed skin surface, or likelihood of ingestion can yield reasonable estimates of human exposure. However, even when environmental data are not available, they can sometimes be modelled. A plume model is often used to estimate likely exposures from airborne releases in which the highest environmental levels (and, therefore, greatest potential for exposure) are downwind. Levels in an explosion are usually modelled as concentric circles.

Another important aspect of the epidemiologic investigation of an industrial disaster is the establishment of baseline monitoring data on the population. Even if no short-term health consequences are expected in the population, data collected as soon as possible after the event are essential in any meaningful evaluation of any subsequent changes in physiological, biochemical, or disease indicators. For acute health effects, baseline data allow the evaluation of the disappearance or persistence of adverse health effects in the population being evaluated. For example, in Bhopal a major concern is permanent pulmonary damage from the MIC. No pulmonary-function testing had been done of the exposed population before the incident; however, evaluation of the changes in pulmonary-function test results of exposed individuals over time will allow assessment of recovery from acute pulmonary effects and development of chronic lung disease. Evaluation of comparison populations may also be necessary for valid inferences about health effects in the exposed groups. This must be considered early in the design of the epidemiologic evaluation to assure that appropriate data are collected. The comparison population may require long-term assessment similar to follow-up assessments of the exposed population.

Epidemiologic studies are also needed to assess factors that affect community compliance with warnings and orders following chemical releases. Warnings and instructions must reach those to whom they are directed. A plant siren may

not be adequate, as was illustrated when methyl isocyanate was released from Institute, West Virginia, and only 5% of residents heard the siren (10). Both inclusion of an explanation in the evacuation order and the use of officials to give orders directly to residents improve cooperation during emergencies (11,12). Studies are also needed on the effects of exposure to actions, e.g., evacuation versus remaining indoors.

Research Recommendations

Most investigations of industrial disasters have focused on causes and environmental impacts. Relatively few epidemiologic studies have been conducted, and surveillance for hazardous materials events is incomplete. Research in the field of industrial disasters should focus on several areas.

Emergency Preparedness

- Data should be collected to identify the types of chemicals being stored and used in the United States and the potentially exposed populations. SARA has provisions for collecting some of these data.
- Quantities and routes of hazardous materials being transported should be determined. These data would provide useful denominators for calculating rates of incidents involving different chemicals and kinds of transport.
- A systematic survey of contingency plans for managing emergencies associated with hazardous material should be conducted. Such a survey would identify areas in which contingency planning is weak or absent.

Emergency Response

- Collection of information on industrial fires, explosions, and chemical releases should be improved. Better systems of surveillance are needed to identify these incidents and their associated morbidity and mortality. When combined with data on chemicals being stored and used, these data will allow determination of rates of incidents by such factors as the chemical or the industrial process. Such rates could be used to target further studies and interventions.
- Collection of information on releases during transportation should be improved, and risk factors for releases during transportation should be studied.
- Evaluation of training and practices for such responders as firefighters should be undertaken. Factors resulting in injury and exposure for such personnel should be evaluated.
- Studies that will lead to recommendations on when to implement evacuations and other emergency-response strategies should be conducted.
- Factors affecting the compliance of individuals in the community with official orders (such as to evacuate) should be evaluated.

Health Outcomes

- More toxicologic and medical information is needed on health effects caused by acute exposures to toxic substances.
- Studies should be conducted to evaluate risk factors for morbidity and mortality associated with industrial incidents.
- For some chemicals, studies should be conducted to relate environmental data to body-burden data and health-outcome data.
- Studies of health outcomes for workers and other exposed persons should be conducted to evaluate long-term and short-term health effects and adverse reproductive outcomes from chemical releases.
- The effects of different medical diagnostic and treatment regimens on health outcomes following exposure to released chemicals should be studied.
- The psychosocial impact of being involved in a chemical release or the emergency response to a release should be evaluated.

Relief Work

- The adequacy of shelters and post-disaster aid should be evaluated.

Summary

The Bhopal incident in India and other recent similar (although less severe) incidents in the United States have focused our attention on the potential for industrial disasters. These disasters are largely preventable through attention to safety features of plants, the use of the least toxic materials possible, zoning regulations, and other actions. Adequate emergency preparedness is necessary for minimizing the adverse public health impact of such an incident. Given the volume of potentially hazardous materials used in our society, incidents will probably continue to occur, making emergency-response activities and training a priority.

Several kinds of data need to be collected in relation to industrial incidents. As recent legislation is beginning to indicate, data identifying the types of chemicals and processes being used in the United States and potentially exposed populations should be available. Post-disaster studies should evaluate health outcomes, impact of emergency-management interventions, and adequacy of relief efforts.

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Fires

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Background to Fire Disasters

Relative Importance of the Problem

Historically, the United States has had a tremendous public health problem associated with fires. Currently, the problem is still of major concern, although its complexity and nature have evolved over time. Contemporary catastrophic fires should be viewed as an unnecessary and preventable problem that certainly deserves the greater attention and efforts of public health professionals.

Each year in the United States, fires result in approximately 5,000 deaths and 300,000 injuries that call for medical treatment (1). Many of these nonfatal injuries require prolonged hospitalization and extensive surgical and medical care. According to some sources, fire disasters cause more loss of life and property damage than all types of natural disasters combined (2).

Unlike many other public health situations, fires are a worse problem in the United States than in other developed countries. Crude fire-associated mortality rates show that the relative risk for persons in the United States is 2-3 times greater (Table 1) than for persons in many other countries. Although these crude rates are not adjusted for population differences with respect to such crucial characteristics as age and sex, the importance of this problem for the United States (and Canada) is certainly clear.

As one type of injury, burn injuries are the second most frequent cause of death in the home, preceded only by falls (3). Burn injuries result in more catastrophic adult fatalities than any other cause (4). In the United States the annual number of adult burn injuries has been estimated at 1 million (5). Estimates of adult rates for burn injuries requiring hospitalization range from 26 to 37 burn injuries/100,000 adults (6-10). With respect to number of years lost by death from specific causes (11), the prevention of a single burn injury fatality results in a greater savings of life than the prevention of death from cancer or cardiovascular disease (12). As shown in Table 1, the fire-associated mortality rate in the United States in 1974 was 2.9 deaths/100,000 persons. This rate has decreased from approximately 4.0 in the 1950s and 1960s (Figure 1). After 1974, the rate increased, and then decreased again to reach a level of approximately 2.3.

Medical Implications of Burn Injuries

One of the most serious insults the human body can experience is a fire-associated burn wound. Burn injuries that require hospitalization are both serious and costly (13). They require more bed-days/patient than any other type of injury (7,14). Furthermore, severe burn injuries are one of the most difficult problems in medicine to treat (12). Patients with burns may need extensive hospitalization with multiple surgical procedures and may be left with lifelong disfigurement and deformity. Severe burn injuries subject both the patient and family to profound psychological and financial stress (15).

The greatest gross effect of burn injuries is the alteration of body-surface appearance. In contrast, the greatest medical impact is the local and systemic physiologic changes. The extent of damage is influenced by many histologic factors. Tissue conductivity greatly affects the absorption rate of thermal energy. Nerves and blood vessels conduct thermal energy at the greatest rate, whereas bone is the tissue most resistant to thermal exchange (16). Connective tissue and muscle conduct energy at intermediate rates (17). A second histologic factor influencing the rate of absorption or dissipation of thermal energy is peripheral circulation (17).

Estimating the extent of a burn injury is necessary for initial triage, for prognosis of long-term morbidity and mortality, and for research purposes. The extent of a burn injury is usually quantitatively expressed as the amount of surface area injured in relation to the total body-surface area. To aid in the estimation of the extent of burn injuries, Berkow first described, in 1924, the percentage of area represented by various body segments (18). These percentages were later modified by Lund and Browder (19) to adjust for age, since the head and neck area of a child constitutes a larger percentage of body-surface area than does that of an adult. A method for quickly determining the body-surface area burned is the "Rule of Nines," devised by Marshall (20). When estimating the total extent of a burn injury, areas with first-degree burns are usually not included (21).

The major pathophysiologic effects of burn injuries involve the cardiovascular system. Burn injuries cause damage to the endothelium, the smaller inner lining of the vessels, which leads to thrombosis. Burn injuries result in dramatic alterations in cardiac function (22). An initial drop in cardiac out-

put occurs, which with extensive burn injuries (> 50% of total body-surface area) approaches 30% of preburn values (23). Cardiac output usually returns to normal values within 36 hours after injury (24,25).

Immediately after a burn injury, capillary permeability in the wound area increases markedly and results in the loss of fluid from the intravascular compartment into the extravascular, extracellular space (26,27). Besides fluid, tremendous amounts of protein (globulin and albumin) are lost into the extravascular space (28-30). Serum leakage through the injured microcirculation results in severe disturbances of body water, electrolytes, serum proteins, and metabolic substrates (31-33). The loss of nutrients through the wound causes a negative nitrogen balance, which contributes to weakness and weight loss (34).

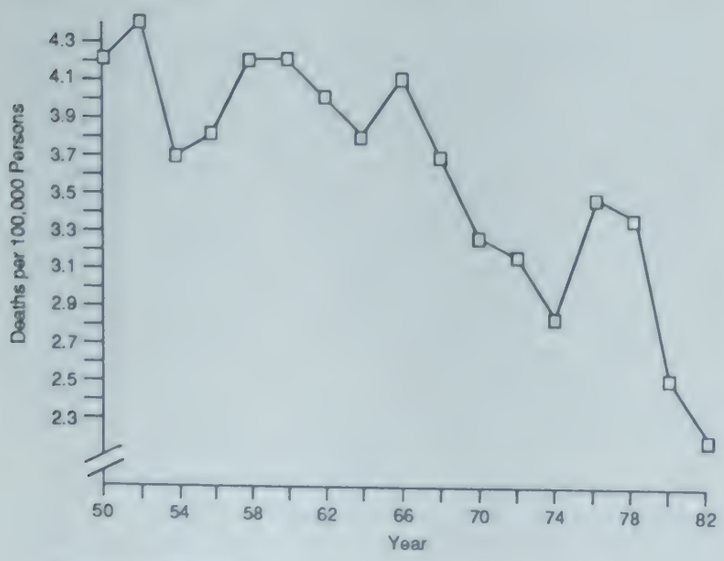
The pathophysiologic changes of burn injuries also involve the pulmonary system. Increased ventilation is usually present and directly proportional to the magnitude of injury (35). With burns of ≥ 40% of total body-surface area, most persons have restrictive pulmonary disease characterized by decreased vital capacity and increased pulmonary resistance. Pulmonary function is greatly affected by concomitant injury caused by toxic combustion products (36-38).

TABLE 1. Fire-associated mortality rates per 100,000 population, by country, 1974

COUNTRY	RATE
Canada	3.6
United States	2.9
Sweden	1.6
Japan	1.5
United Kingdom	1.5
France	1.5
Australia	1.5
West Germany	0.9
Switzerland	0.7

Source: Reference 1.

FIGURE 1. Fire-associated mortality rates, by year, United States, 1950-1982



Source: Reference 1.

Historical Perspective on Fire Disasters

Fire disasters can be attributed to extremely varied causes. They may accompany natural disasters such as earthquakes and volcanic eruptions. Sources of ignition for fire disasters have included lightning, human carelessness, arson, and malfunctioning equipment. These disasters have occurred above the ground (in tall buildings and on planes), on the ground, and below the ground (in mines and caves). Sometimes they occur in circumstances that are unexpected or unpredictable. For example, several fire disasters have involved the spraying of flaming liquids into crowds; in 1955, this scenario resulted in the deaths of 80 people who were watching the LeMans Grand Prix race. The mixture of causal patterns has varied with time, and one can hardly say that contemporary America has the same risks that it had 30, 50, or 100 years ago. A basic understanding of how these risks have changed assists in identifying preventive measures that have apparently been efficacious in the past or may need to be assessed in the future.

Seven descriptive categories of fire disasters, along with selected examples, are shown in Table 2 (39). The first category pertains to disasters resulting from forest fires. The three examples provided occurred either before or during the early 1900s and involved three different states. During this period, information dissemination and warning systems along with fire fighting and control capabilities did not compare with those available today. These types of disasters have evolved to the point that they have far greater impact on the environment than on the public health of surrounding communities. Each year such fires destroy thousands of acres of valuable grass and timberland, while their impact on morbidity and mortality is minimal. Examples of this modified impact of forest fires include the 1947 Maine fire (16 deaths; 1,200 structures damaged; and 206,000 acres [83,400 hectares] of timber and scenic forest destroyed); the 1964 Cayote, California, fire (one death; 188 structures damaged; and 175,000 acres of watershed land destroyed); and the 1977 Santa Barbara, California, fire (no deaths, but 250 structures damaged, along with 800 acres of watershed land destroyed).

Tangentially related but basically different are fire storms—both naturally occurring and human generated. These natural storms develop from forest fires. They result in a convection plume consisting of hot gases that cause air to be drawn inward at the base. This wind then begins to rotate and forms a fire-induced cyclone that, like a tornado, has counterclockwise winds in the Northern Hemisphere. Apparently, the worst natural fire storm occurred at Peshtigo, Wisconsin, in 1871. It burned over 2,000 miles² (5,180 km²) of forest and killed approximately 2,300 people. Near Sundance, Ohio, in 1967, a fire storm had surface winds of 50 mph (80 km/hour), peak winds of 120 mph; it lasted for 9 hours. This fire storm destroyed 70 miles² of land. However, the incidence of natural fire storms is low enough that the adverse public health impact is small.

Human-generated fire storms resulted from incendiary bombing during World War II. In Hamburg, Germany, on February 27, 1943, the Allied Air Forces dropped bombs that caused a fire storm with winds up to 100 mph, destroyed 3.2 miles² of city, and killed 21,000 residents. In Dresden, Germany, on February 13-14, 1945, bombs induced a fire storm that had surface winds up to 80 mph, burned 4.6 miles² of

TABLE 2. Selected fire disasters, by category, date, and associated mortality, United States

Category		Date	Number of fatalities
Forests:			
	Michigan and Wisconsin	1871	1,000
	Minnesota	1894	894
	Minnesota and Wisconsin	1918	1,000
Cities:			
	Chicago	1871	766
	Peshtigo, Wisconsin	1871	800
	San Francisco	1906	1,188
	Chelsea, Massachusetts	1908	18
Ships:			
	New York harbor	1904	1,000
	Rhode Island coast	1954	103
Hotels:			
	Winecoff (Atlanta)	1946	119
	LaSalle (Chicago)	1946	61
	MGM Grand (Las Vegas)	1980	84
	Hilton (Las Vegas)	1980	8
	Stouffers Inn (New York)	1981	26
Places of entertainment:			
	Theater (Chicago)	1903	602
	Dance Hall (Mississippi)	1940	207
	Nightclub (Massachusetts)	1942	492
	Circus (Connecticut)	1944	163
	Supper Club (Kentucky)	1977	164
Health-Care Facilities:			
	Hospital (Oklahoma)	1918	38
	Nursing home (Missouri)	1957	72
	Hospital (Connecticut)	1961	16
	Nursing home (Ohio)	1963	63
	Nursing home (Ohio)	1970	31
Schools:			
	Collinwood, Ohio	1908	161
	Chicago, Illinois	1958	93

Source: References *1,39*.

city, and killed 135,000 persons. On March 20, 1945, an incendiary attack on Tokyo resulted in a fire storm that killed 84,000 persons.

Like forest-fire disasters, U.S. citywide conflagrations were most devastating before or during the early 1900s. The sources of ignition for this type of fire disaster were both human generated (Chicago fire) and natural (San Francisco fire). The source of combustion was frequently wooden structures crowded on small land-surface areas. The contemporary risk of this category of fire disaster has been minimized by the development and enforcement of building codes, promulgation of standards to ensure the compatibility of all fire equipment with water supplies, and the regulation of building construction with respect to combustible materials.

Regarding the last category, considerable effort has been expended in the design and construction of "fireproof" buildings. Progress has been due in part to the evaluation of unsuccessful approaches. For example, when the Crystal Palace was built in Manhattan in 1853, it was considered to be fireproof since its frame was iron and its walls and roof were glass. Although iron and glass are not combustible, the contents of this building were. In 1858, a fire that began in the interior caused the entire building to collapse. The heat from the fire melted the crucial iron structural supports. The lesson learned from this incident was to have concern for combustible materials within buildings and to insulate structural steel so that it can not reach its 500 C melting point.

The third category of historical fire disasters consists of places in which groups of healthy persons reside on a temporary basis, for example, on ships and in hotels. In recent times, building codes have increased safety in such places by establishing criteria for interior passages, stairwells, and exits. These criteria are designed to prevent passageways and

stairwells from becoming chimneys or disseminators for fires and to ensure that people have ample means of escape. Public health problems can result when these codes are not followed. In the United States from 1934 to 1961, 130 hotel fires killed 1,204 people (1). In November 1984, the Las Vegas MGM Grand Hotel fire killed 84 people. Investigation of this disaster showed that three of the four stairwells and their access panels did not comply with codes for 2-hour fire-rated construction (1). The fire and products of combustion that killed people spread through these stairwells.

Places of entertainment present special problems for the enforcement of fire codes. In such locations, large numbers of persons are crowded into unfamiliar and enclosed spaces. Either the exits (malfunction or inadequate number) or the furnishings and decorations (large quantities of flammable materials) may be problems. Perhaps the most famous fire disaster in this category was the Coconut Grove nightclub fire in Boston in November 1942. In this incident, most exits were either locked or they malfunctioned. Approximately half of the crowd—492 persons—died, and many others sustained serious burns.

For the most part, the potential for fire disasters in places in which people temporarily reside or seek entertainment involves the exposure of persons who have unimpaired physical and mental capabilities. Locations such as health-care facilities and schools, in which the exposed populations depend upon the providers for safety and well-being, are associated with even higher risks. These risks appear to be reduced when the occupied buildings are designed for the specific purposes intended, evacuation plans are developed and practiced, and engineering controls (e.g., fire doors and sprinklers) are present.

Epidemiology of Fire Disasters

Working Case Definition

The literature suggests many criteria for defining disasters. Some of these include cause, duration of occurrence, extent of damage, and number of casualties. With respect to the last criterion, suggested quantitative values are often provided. For example, one source (40) states that for an incident to be classified as a disaster, it must cause at least 25-100 casualties (injuries and deaths). However, agencies such as the Metropolitan Life Insurance Company (MLIC) and the Occupational Safety and Health Administration (OSHA) consider a catastrophe (disaster) to be an event that causes at least five casualties—either deaths (MLIC) or deaths plus hospitalizations (OSHA). This small number of victims from a single event is probably not perceived to be a disaster by many public health professionals. However, it is essential—both for the review contained in this chapter and the future prevention of fire disasters—that the definition of fire disaster include situations with only a few fatalities.

There are very few studies focusing on the epidemiology of fire disasters. Salient characteristics of selected, representative studies pertaining to burn injuries are shown in Table 3 (41-49). All of these investigations have been cross-sectional studies, and all but three are hospital-based case series. Consequently, they focus upon the more severe burn injuries. The number of cases per report ranges from 100 to 2,927. Two important trends are demonstrated in these reports. First, all but two of the studies are historical reviews and depend on data from medical records. Investigations of this kind are limited in types of variables that can be examined. Second, and more importantly, none of these authors distinguished between cases that did and did not result from disasters. Therefore, most available descriptive information about fire disasters is limited to surveillance statistics maintained by agencies such as MLIC and the National Fire Protection Agency (NFPA).

If the MLIC definition of disaster is used, fires represent the largest source of disasters in the United States (2). The NFPA appears to evaluate fire disasters by examining multiple-death fires that involve at least three fatalities. NFPA data demonstrate the public health importance of these events. In 1983, in the United States, 2,326,500 fires resulted in 5,978 deaths. Multiple-death fires accounted for only 0.01% of all fires but led to 16.4% of all fire-associated deaths (50).

Although distinguishing between definitions based upon events resulting in at least three deaths versus at least five deaths may appear trivial, it is important from a public health perspective. NFPA data shown in Table 4 indicate that in the United States in 1980-1984, 1,391 fire disasters (at least three deaths) caused 5,639 deaths, or an average of 4.1 deaths/incident. For each year in this 5-year period, the average number of deaths/incident was less than five. More extensive analysis shows that approximately 80% of these incidents each led to three or four deaths (Table 5). Fires that caused at least 10 fatalities represented only 1.9% of the multiple-death fires. Any definition of and research on fire disasters that focus on incidents involving at least 10 deaths—or even at least five deaths—fails to include the vast majority of events that impact mortality and represent the greatest public health risk. Therefore, the working definition of a fire disaster in this chapter is any fire that causes at least three deaths.

Severity of Injury: Implications for Mortality and Morbidity

Public health professionals should not equate the **severity** with the **extent** of injury from a fire disaster. The extent of a burn injury is denoted by the total area of body surface that sustained second- or third-degree burns. In contrast, the severity is determined not only by the extent of injury but also by anatomic location, age, physical condition, presence of preexisting disease, and presence of concomitant injuries (51). In addition, after adjustment for confounding factors, length of hospital stay has been used as an approximation for severity of some burn injuries (52-56).

For various critical anatomical areas of the body, burn injuries result in loss of function (sensory or motor or both) and disfigurement that must be considered serious even though the extent of the injuries may be small. These critical anatomic areas include the face, hands, feet, external genitalia, neck, and joint surfaces. Persons with preexisting renal, cardiovascular, or pulmonary disease cannot tolerate burn injuries as well as those without such disease. For persons with occlusive vascular disease, burn injuries to the lower extremities (especially the feet) are particularly serious. For adults with peripheral arteriosclerosis, gangrene requiring amputation is not uncommon after burn injuries to the feet or legs.

TABLE 3. Characteristics of selected epidemiologic studies of burn injuries

Year(s) of study	Number of cases	Subjects*		Data collection†		Source:
		I	I+-O	RR	FU	(Reference #)
1970	100	X			X	41
1972-1973	155	X		X		42
1970-1975	411	X			X	15
1965-1974	386	X		X		43
1972-1975	1,049		X	X		44
1974	380		X	X		45
1974	2,862	X		X		6
1974-1975	1,165	X		X		8
1974-1975	2,927	X		X		46
1973-1976	2,729		X	X		47
1976	200	X		X		48
1974-1977	822	X		X		49

NOTE: None of these studies distinguished between burn injuries resulting from fire disasters (fires that cause at least three deaths) and those resulting from fires not classified as disasters.

*I = inpatients; I+O = inpatients and outpatients.
†RR = record review; FU = follow up.

TABLE 4. Incidence of fire disasters and associated mortality, United States, 1980-1984

TOTAL Year	Number of fire disasters	Number of deaths	Average number of deaths/disaster
1980	326	1,356	4.2
1981	296	1,179	4.0
1982	266	1,111	4.2
1983	259	986	3.8
1984	244	1,007	4.1
TOTAL	**1,391**	**5,639**	**4.1**

Source: Reference *50*.

Burn injuries may lead to new cardiovascular or pulmonary disease. The most common types of pulmonary disease include pneumonia and atelectasis. Ophthalmic (57), renal (58), and neurologic (59) disease may develop after some types of burn injuries.

Two major types of concomitant injuries may result from catastrophic events that cause burn injuries: inhalation injuries and fractures. An inhalation injury is caused by breathing in noxious gases and is the most serious concomitant injury (60). Smoke from some fires contains nitrogen dioxide and sulfur dioxide, which may cause bronchiolitis (61), alveolitis (62), and broncospasm (63). Clinical features of inhalation injury include nasal-membrane irritation, pharyngeal edema, hoarseness, and bronchorrhea. Fractures may also compound the burn injury in accordance with their severity. The presence of fractures in association with burns complicates treatment and prognosis for both the burn injury and fracture (64,65).

Inhalation injury is even more important in the context of fire-disaster-related mortality. Most victims succumb to the asphyxiating effect of carbon monoxide long before the flames or heat affect them directly (66,67). Also, carbon dioxide poisoning or oxygen deficiency may play a role (68). During fire disasters within buildings, the confines of the structure assist in retaining and concentrating the toxic combustion products and smoke from the fire (69,70). A smoldering mattress or sofa in a standard-size room can produce lethal levels of carbon monoxide in as little as 30 seconds (71).

TABLE 5. Frequency and percentage distribution of fire disasters, by number of deaths/fire, United States, 1980-1984

Number of deaths/disaster	Number of fire disasters	Percentage of fire disasters
3	777	55.8
4	336	24.1
5	135	9.7
6	57	4.1
7	39	2.8
8	18	1.3
9	4	0.3
≥10	26	1.9
TOTAL	**1,391**	**100.0**

Source: Reference *50*.

It is also important to realize that fire-associated mortality may not result directly from the fire or its products. First, some natural deaths may cause a fire to start after the

death (71)—e.g., a fatal cardiac collapse while smoking, while using matches or a lighter, or while being near an open flame (candle or stove). Although fire fighters are one of the occupational groups at greatest risk of dying on the job, the direct effects of fires are not the greatest killers (72). The most prevalent causes of death while responding to fires are heart attacks and vehicular accidents (72).

Contemporary Fire Disasters: Place of Occurrence

In the contemporary world, fire involving heat generation from burning fuel is converted into electrical/mechanical energy and does practically all the work of industrialized countries. Ironically, the current public health risks of fire disasters in the United States occur away from places in which fire does the work. Furthermore, the relative importance of many locations involved in historical fire disasters (Table 2) has decreased, and a different high-risk location for fire disasters has emerged that deserves the greatest public health concern and emphasis.

According to a Metropolitan Life Insurance Company study, fires accounted for 31.2% of all disasters in the United States in the period 1941-1975 (Table 6). Furthermore, fires accounted for 26.9% of all disaster-associated mortality. In these fire disasters, 68.3% of the incidents and 47.1% of the associated deaths occurred in houses or apartments. Temporary public residences (hotels and boarding houses), treatment centers (nursing homes and hospitals), and public places accounted for only 7.4%, 4.3%, and 0.9%, respectively, of all fire disasters.

TABLE 6. Civilian disasters and associated deaths, by type of disaster, United States, 1941-1975

Type of disaster	Number of incidents	Number of deaths
Fire and explosion	1,369	12,128
Houses, apartments	935	5,716
Hotels, boarding houses	101	1,072
Hospitals, nursing homes	59	861
Public places	12	835
Other	262	3,644
Motor vehicle	1,659	10,516
Air transportation	471	7,756
Water transportation	225	2,226
Railroad	78	1,342
Weather phenomenon	335	8,279
Mines and quarries	94	1,612
All other	162	1,252
TOTAL	**4,393**	**45,117**

Source: Reference 4

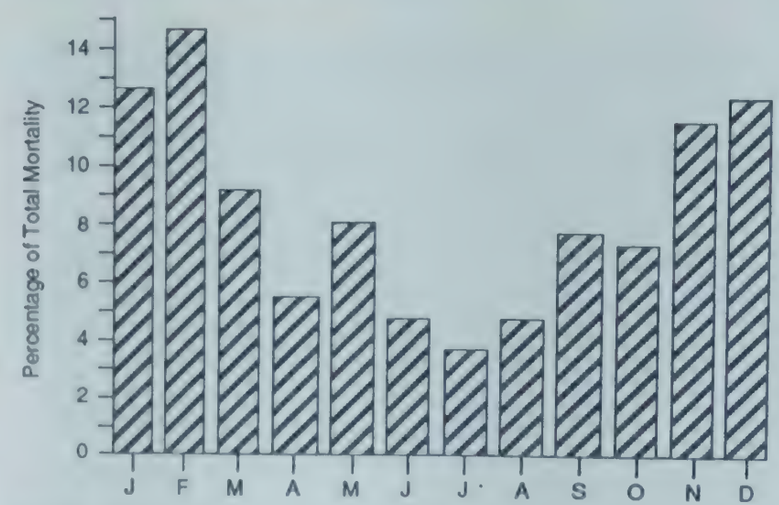
More recent data from the NFPA show that in the United States in 1980-1984, 87.5% of all fire disasters and 83.5% of all associated deaths occurred in residential properties (50). For 1984, 67.2% of all residential fire disasters occurred in one- or two-family dwellings (excluding mobile homes), 16.3% occurred in apartments, 10.9% occurred in mobile homes, and 6.6% occurred in rooming or lodging facilities. Therefore, from a public health perspective, any focus on fire disasters should emphasis occurrences involving single-family residences or duplexes.

Environmental and Human Descriptive Characteristics

NFPA data for the United States fire disaster experience in the period 1980-1984 have been reported according to four broad regions—Northeast, North Central, South, and West. For this reference period, fire disaster death rates (per 1 million persons) were 6.2, 5.3, 5.6, and 3.0, respectively, for these regions. Overall and for each year within this 5-year period, residents of the West had the lowest rate, while those of the Northeast had the highest rate. It is extremely difficult to draw any conclusions about these experiences because of unknown contributions made by many different factors—including climate, socioeconomic status, amount of urbanization, population density, and the age and construction of buildings.

Available information pertaining to the 1984 monthly distribution of fire disaster mortality is shown in Figure 2. Published statistics do not include information about the monthly distribution of incidents. Therefore, the observed differences among months may result from variation among the numbers of incidents and/or the average number of deaths per incident. Months associated with cold weather account for the largest percentages of deaths. December, January, and February accounted for 39.3% of all mortality. Further analysis by the NFPA showed that at least 25% of all deaths during these 3 months were associated with some type of heating equipment. However, these months still appear to have an excess mortality above that attributable to heating equipment.

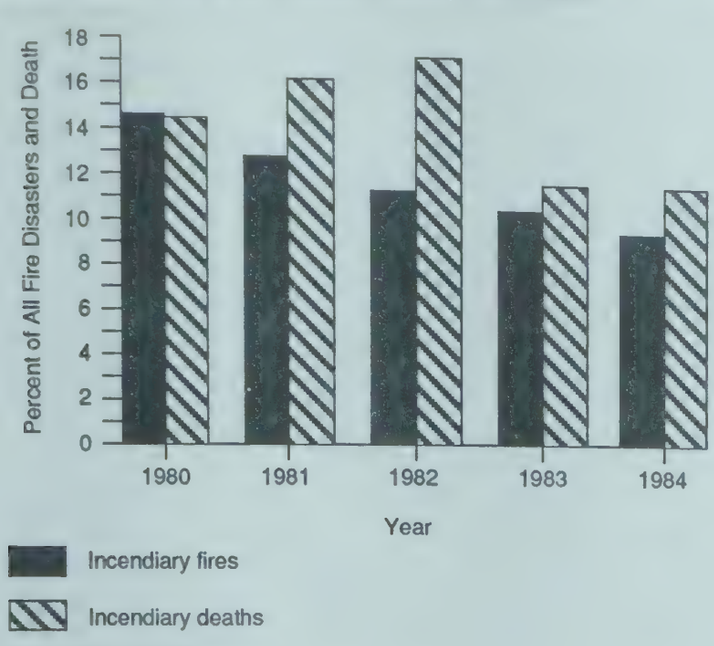
FIGURE 2. Percentage distribution of fire-disaster mortality, by month, United States, 1984



Source: Reference 50

With reference to accidental versus nonaccidental causes, NFPA data shown in Figure 3 indicate that for 1980-1984, incendiary (deliberately set) fires accounted for 12% of all fire disasters, with an annual range of 9.4% (1984) to 14.7% (1980). This annual percentage distribution implies a decreasing importance of nonaccidental causes for fire disasters. The data on annual deaths associated with incendiary fire disasters show that for the same period, 14.3% of all fire

FIGURE 3. Percentage distribution of incendiary fire disasters and associated mortality, United States, 1980-1984



Source: Reference *50.*

disaster deaths were associated with this cause, with a range of 11.5% (1983, 1984) to 17.1% (1982). These data indicate that incendiary fire disasters tend to result in larger numbers of fatalities. There appear to be two explanations for this trend. First, incendiary fires are more likely to take place in nonresidential locations containing more people. In 1980-1984, incendiary fires accounted for 32% of all nonresidential fire disasters and only 16.2% of all residential fire disasters. Second, other NFPA data show that 44.8% of all incendiary and 'suspicious" fire disasters originated in egress areas. Blocked exits apparently create a greater risk that persons inside will die.

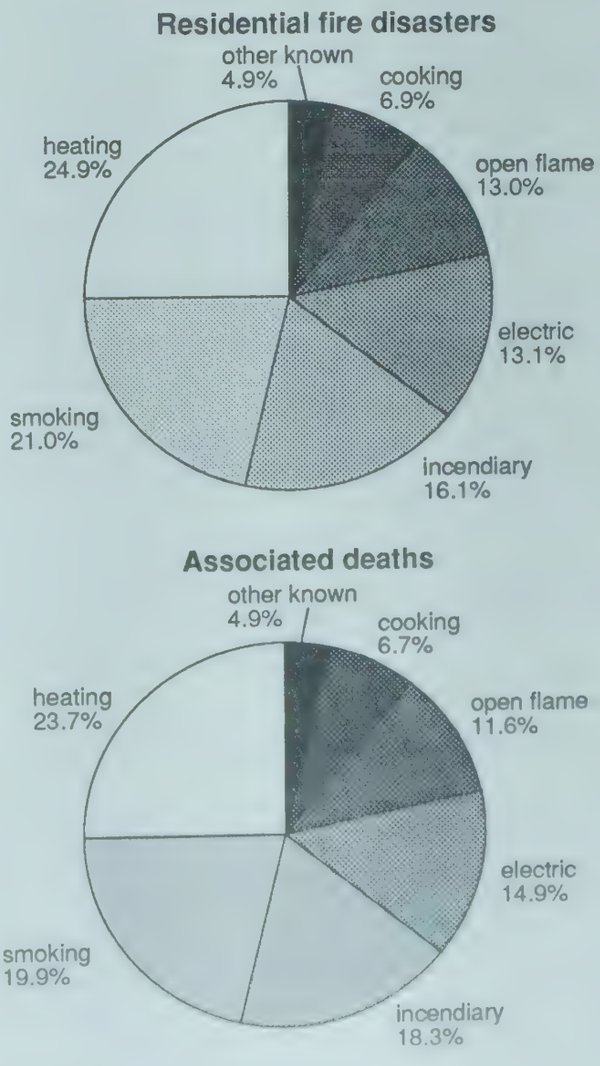
It has already been shown that in contemporary America the vast majority of all fire disasters and associated mortality have occurred in residential properties, mainly in single-family homes and duplexes. A recent control strategy designed to minimize the risks of being in such locations during fires is the installation of smoke detectors. In 1984, 74.1% of both residential fire disasters and associated mortality occurred in dwellings with no smoke detectors. These statistics do not account for instances in which detectors are present but improperly installed or maintained. Although apparently no comparable denominator data are available, these percentages appear spuriously large. The importance of this control strategy is perhaps best supported by statistics pertaining to the time of day at which these disasters occur. In 1984, 66.8% of all residential fire disasters occurred between 12:00 midnight and 8:00 a.m. In conclusion, given this trend in the time of occurrence and the greater influence of smoke and combustion products compared with the flames on the risk of mortality, proper utilization of smoke detectors seems to be an essential public health prevention strategy.

NFPA statistics pertaining to sources of ignition of residential fire disasters and mortality are shown in Figure 4. The largest source of ignition for both incidents and deaths has been heating equipment. For this category, > 90% involved auxiliary heating equipment rather than principal sources. In 1980-1984, of all residential fire disasters that involved auxiliary heaters, 41.1% involved fixed heaters (wall heaters, wood stoves, etc.; 38.9%, portable heaters; 8.2%, chimneys; 5.6%, fireplaces; and 6.3%, connections.

The second leading source of ignition for residential fire disasters is smoking. For this category, the incident distribution by specific location includes the living room (72.4% of the incidents and 73.4% of the deaths), bedroom (19.9% of the incidents and 19.2% of the deaths), and the kitchen (4.6% of the incidents and 4.7% of the deaths). Since most such incidents occur late at night or early in the morning, a likely scenario appears to be that persons fall asleep while smoking in the living room or bedroom.

The third and fourth leading categories of ignition in residential fires are arson and electricity, respectively. Both sources account for more deaths per incident than do the other sources of ignition. Many points addressed in the discussion of incendiary fires would be applicable here as well. Fire disasters due to arson and electricity in residential locations may more frequently involve a) elimination of the capability to exit or b) multiple sites of ignition. The greater contribution to mortality by electricity may involve fixed wiring located in concealed spaces. Consequently, when such fires start, they may go undetected longer and place residents at greater danger.

FIGURE 4. Residential fire disasters and mortality, by source of ignition, United States, 1980-1984



Source: Reference *50*

The fifth leading category of ignition is open flames. For this category, leading sources of open flames are matches (39.9%), lighters (25.7%), and candles (22.3%). Probably the most important statistic is the percentage of all residential fire disasters caused by children using (playing with) matches. Approximately 11% of all residential fire disasters begin this way, an important but much smaller contribution than has been traditionally perceived by the public and by many public health professionals.

Of the host characteristics that have been assessed, age appears to be an important risk factor. In 1984, 53% of all persons killed in residential fire disasters were < 15 years of age, compared with only 22.2% of the general population at risk (50). Persons in this age group may be either too young to react on their own or may react improperly because of insufficient knowledge of safe behavior. Interestingly, elderly persons (> 65 years of age) accounted for only 5.8% deaths in all residential fire disasters and did not represent a high-risk group. However, this may merely be a consequence of either living singly, in couples, or in nursing homes which, by definition, would not place them at risk from residential fire disasters.

Although not specific to catastrophic fires alone, an important host characteristic for sustaining burn injuries is any predisposing medical factor. In one study of 500 hospitalized adult burn patients, the vague term "poor judgment" was implicated in persons having sustained burn injuries (73). Elderly adults are more prone to severe burn injuries than are younger adults, possibly because of more limited defensive and reactive capabilities (74). Data from three studies of adult burn injuries that examined the contributory influence of predisposing medical factors are shown in Table 7 (74, 75). Approximately one-fourth of all adults who sustained burn injuries had some type of predisposing factor. These studies show that the two most important factors are alcoholism and epilepsy. Other studies have shown that 5%-10% of all adult burn injuries are sustained by individuals subject to epileptic seizures (77-82), and that alcoholism also plays a prominent role in the occurrence of adult burn injuries (83,84).

Prevention Strategies

Epidemiology: Surveillance and Research

Epidemiology can play an important role in preventing or mitigating the adverse public health impact of fire disasters. To date, this role has been extremely limited and used primarily to discern the differences in efficacy among various clinical strategies for treating persons with serious burn injuries. Although associated epidemiologic activities have focused largely on generic burn injuries or burn injuries associated with a specific type of location (workplace, home, recreation, etc.), the need for separating burn injuries by severity of incident (disaster versus nondisaster) has apparently not been totally recognized.

As mentioned earlier, few descriptive or analytic data on the adverse public health impact of fire disasters are available. Data are basically limited to surveillance statistics maintained and published by a few agencies and gathered from case reports of fire disasters. Limitations in these data include the lack of denominator data needed to draw more valid conclusions about risk factors, insufficient description of associated morbidity, and failure to distinguish between characteristics and risk factors of fire disasters that are unique versus those that are similar for all types of fires.

A full spectrum of epidemiologic activities encompassing both surveillance and research would almost certainly assist in the further prevention or mitigation of fire-associated mortality and morbidity.

Engineering and Legal Controls

Many people may not be aware that the general acceptance of skyscrapers and high-rise buildings in the United States has resulted in part from the identification, establishment, and enforcement of building codes. The concept and adoption of building codes in this country can hardly be considered a new prevention strategy.

TABLE 7. Frequency and percentage distribution of predisposing medical factors causally associated with burn injuries sustained by adults, three studies

Predisposing medical factor	Pegg et al. (15) (n=411)		MacLeod (74) (n=723)		Pegg (75) (n=170)	
	Number	Percentage*	Number	Percentage*	Number	Percentage*
Alcoholism	26	6.3	65	9.0	12	7.1
Epilepsy	11	2.7	27	3.7	9	5.3
Psychosis, neurosis	8	1.9	19	2.6	8	4.7
Drugs	6	1.5	17	2.4	5	2.9
Suicide	11	2.7	4	0.6	4	2.4
Mental defect	9	2.2	4	0.6	3	1.8
Syncope	NR†	NR†	8	1.1	4	2.4
Diabetes	7	1.7	4	0.6	2	1.2
Other	20	4.9	18	2.5	1	0.6
TOTAL	**98**	**23.9**	**166**	**23.1**	**48**	**28.4**

*Expressed as percentage of total burn injuries in the study
†NR = Not reported

Source: References *15, 75, 76*

The first U.S. building codes were implemented by municipalities in the late 19th century. These early codes addressed the prevention of conflagration and were designed to minimize the risks that fires would spread to neighboring buildings. These codes provided specifications for roofing and exterior materials and characteristics (such as thickness and fire resistance) of common walls.

At the initiation of insurance companies, the National Fire Protection Agency (NFPA) was established in 1896. This organization has played a vital role in augmenting building codes and regulations. For example, codes have been developed for addressing such issues as fire-wall performance, separation between freestanding structures, and storage of combustible materials.

The evolution of codes is in some ways associated with the evolution of fire disasters in this country. The early threats of fire disaster focused around urban conflagration. Today, with peacetime conditions, this type of fire disaster represents little public health threat. The chief concerns today deal with fire inside—rather than among—buildings that are primarily residential. For the threat of fires within commercial buildings, codes provide for public safety by detailing stipulations for interior passages, stairwells, and doors. These codes provide for protective strategies involving containment of fire and/or evacuation of people.

Although the greatest contemporary risk of fire disaster involves single-family residences and duplexes, the thrust of building codes for these structures is different in several ways. First, these codes tend to involve less expensive strategies since individuals rather than businesses must bear the cost. Second, the containment of fire within certain areas of the structure is not a viable approach because of the size of residences. Third, because of the lack of access for inspection and the number of residential buildings, codes that require routine inspection of visible structures are not practical. Consequently, residential building codes have focused on structures not seen or difficult to correct—e.g., the design of chimneys and the placement of electrical circuits and wiring. These codes are enforced when the building is being constructed (or remodeled).

A recent engineering control that increases the length of warning time and represents a fairly inexpensive investment that can be made at any time is the smoke detector. The data presented, though limited, support the greater need for this control to be utilized within residential homes.

Mitigation Response and Suppression

In the United States, the training of fire fighters and the fighting of fires are old and established practices. The earliest response to fires consisted of ad hoc bucket brigades. The first fire-fighting company of trained individuals was founded in the 1730s by Benjamin Franklin (1).

The development of fire-fighting techniques for control and prevention of fires has augmented codes to minimize the risk of fire for whole sections of cities and communities. An important but often unrecognized function of fire departments is the inspection of buildings to enforce compliance with codes that govern construction, maintenance, and occupancy. However, the focus of most activity associated with this function is on nonresidential structures. Also, some of the fire-fighting strategies for large buildings are not suited for single-family dwellings and duplexes. For example, a house fire cannot purposely be allowed to burn beyond the room in which it started since there is no realistic way to contain it before it envelops the entire structure.

However, the ability of fire departments to reach any portion of their catchment area within minutes of receiving a fire alarm has minimized the risk of injury to persons and damage to property once residential fires have started and been discovered. Each year in the United States, fire departments respond to approximately 1 million residential fires (1). Given the amount of resources committed to and the realized accomplishments of this prevention strategy, additional substantial improvements in fire-fighting strategy that would further impact on the public health implications of fire disasters are not very likely.

Medical Treatment and Rehabilitation

Extensive clinical and epidemiologic work has focused on the triage, management, and rehabilitation of victims of fire disasters (85-89). Also, the emergency-medical-care components of disaster plans have been successfully implemented, as was the case when 1,700 fire victims from the MGM Grand Hotel fire were cared for (90). Prior discussions about the medical implications and severity of burn injuries are only slightly indicative of the tremendous amount of scientific knowledge currently available concerning the medical consequences of and treatment for burn injuries. Burn units in hospitals or entire hospitals devoted to burns operate throughout the United States. Surgical and medical treatment has not only maximized the likelihood of survival but also the aesthetic and functional potential for victims of serious burns.

As a means of tertiary prevention, medical treatment and rehabilitation have reached a plateau in ensuring survivorship and reducing morbidity associated with fire disasters. Further, significant reductions in fire disaster morbidity and mortality depend heavily on primary prevention approaches. These approaches entail activities directed during the pre-event phase of the disaster to prevent fires, reduce human exposure to the thermal energy of fire, or decrease the susceptibility of humans to injury. Primary prevention approaches should not only minimize public health impacts but may also improve adverse economic and social conditions associated with fire disasters. To realize future reductions in the public health impacts of fire disasters, it seems more efficacious to expend any additional resources on primary prevention strategies, such as public education and awareness.

Public Awareness and Education

As with any public health problem, once risk factors and prevention strategies have been identified and accepted by research and public health professionals, any reduction in the magnitude of the problem depends upon the awareness and education of the public at risk. Certainly, fire disasters are no exception. In fact, the need for public awareness and education may be more important for fire disasters than for other public health problems if one considers the size of the population at risk and the incidence of fires. Most of the U.S. population lives in single-family homes or duplexes, and 1 million fires occur in such residencies each year.

Persons need to understand the risk of fire disasters associated with their residences. Since 1980, major fires in hotels that received national attention have sensitized the portion of the public who regularly use hotels to the need to be knowledgeable about appropriate means of egress and reaction during fires. However, the entire adult public should be able to apply the same basic knowledge to fires in their homes. Children who are old enough should be trained by their parents and teachers about what to do if there is a fire, and plans should be made to take care of young children. Families should hold rehearsals to help instill appropriate actions. Adults should recognize the risks that auxiliary heaters and cigarettes pose as sources of ignition for residential fires. Efforts should be made to install and maintain smoke detectors on each level of the home. These are just a few of many examples of the ways that the public needs to become aware of and educated about fire disasters.

Critical Gaps in Knowledge

Public health professionals may lack knowledge concerning the characteristics and public health impact of fire disasters in the United States. Their concept of a fire disaster should be adjusted to reflect a large frequency of incidents, each of which involves only a few deaths and usually occurs in the home. Although quite different from what is usually perceived to be a disaster, this kind of incident represents the contemporary fire disaster problem.

Current data for fires and fire disasters are inadequate—in terms of completeness, accuracy, and comparability (2). Sources of data include the National Center for Health Statistics, the National Fire Protection Agency, various members of the insurance industry, the National Fire Protection and Control Administration, the National Household Fire Survey, and reports from State Fire Marshals. Statistics published by various sources may differ because of different objectives, assumptions, and methods of collection and analysis. Much of the data in this chapter represents statistics published by the National Fire Protection Agency and appear to be the most comprehensive and detailed information available. However, it is uncertain how they vary from data collected by other sources.

Very little information is available on morbidity associated with fire disasters. Most available information does not cover nonfatal injuries. It is presumed that—as with other injury scenarios—numerous serious burn injuries and even more minor burn injuries occur for every fatality associated with a fire disaster. Given the potential for the tremendous burden of hospitalization for burn injuries on medical, economic, and social systems, sufficient public health knowledge about these injuries is essential.

Detailed information for risk assessment is lacking. Available data for fire disasters are mainly limited to surveillance data based on the aggregation of individual case reports. With the lack of denominator data and detailed characteristics, only crude conclusions about risk can be drawn. Furthermore, existing data make it extremely difficult to determine the efficacy of various types of prevention strategies.

Current literature does not directly address differences and similarities between fire disasters and all fire incidents. It is helpful to understand which characteristics of fire disasters are unique and which are similar for all types of fires. This understanding would assist in setting priorities for research needs and detailed preventive strategies.

Fire disasters may often be thought to result from a single causal factor. Some examples exist in which several factors are considered jointly to better understand the disaster scenario and the relative contributions of individual factors. Professionals have used stratified analyses, but have encountered problems because of the necessity of using small numbers. In some instances in the literature even the most frequent pattern of factors is relatively unimportant because it represents only a small percentage of fires. An increased knowledge of the comparability of databases is essential in order to facilitate aggregation so that larger numbers may be obtained for multivariate statistical analyses.

A review of the literature did not provide a complete appreciation for the operating assumptions adopted by groups addressing either the prevention or suppression of fires. With public health implications, there appear to be two different assumptions—the goal may be to prevent the initiation of the fire, or the goal may be to control the fire or evacuate the people. Such knowledge would be helpful in developing a thorough understanding of progress to date and in anticipating future needs and advances of these groups.

Most deaths from fire disasters result from the inhalation of combustion materials produced by the fire. Some of the related fundamental knowledge needed for prevention includes how gases are produced by and distributed during a fire disaster and how best to detect and warn potential victims about the presence of such gases. General gas processes in a fire disaster need to be better understood as they pertain to ignition, smoldering combustion, early stages and spread of flaming combustion, and distribution dynamics in rooms and corridors.

Most building codes in the United States focus on nonresidential buildings, although existing data show that the contemporary problem of fire disasters is with residential structures. More information is needed concerning the appropriateness and effectiveness of augmenting existing residential building codes.

It has already been mentioned that the threat of urban conflagration in peacetime is not a major public health problem. However, there is a critical knowledge gap concerning the potential new threat of suburban conflagration in some states. For example, to minimize the potential of erosion in some desert states, the chaparral has been allowed to remain close to walls or yards in hillside residential and commercial developments. This practice may increase the risk of conflagration from brush fires.

As with most public health problems in this country, state and federal efforts to prevent fire disasters augment those activities at the local level. Currently, there is a lack of detailed knowledge about strength, success, and needs of local efforts.

A key prevention strategy appears to be public awareness and education. Yet, the extent of the general public's basic understanding of fire disasters is unclear. More knowledge about baseline levels is needed, especially for high-risk parts of the country.

Public Health and Research Recommendations

The following activities are recommended in efforts to improve the identification and efficacy of prevention strategies designed to prevent or mitigate public health impacts of fire disasters:

1. The public and health professionals should become better educated about the true, insidious nature of contemporary fire disasters.

2. Appropriate agencies and public health professionals should develop greater concern for and focus more efforts on morbidity from fire disasters.

3. Efforts should be undertaken to maximize uniformity and comparability of data sources.

4. Existing data systems should be modified or new systems developed as appropriate to provide descriptive data with more detailed characteristics of human and environmental factors and applicable denominator data.

5. Since most information about characteristics and the public health impact of fire disasters derives from surveillance, efforts are needed to design and conduct epidemiologic studies that provide analytical data about risk factors.

6. Through consultation with appropriate fire prevention agencies, the need for specific epidemiologic studies should be determined and supported. For example, population surveys of level of education or safety practices are appropriate. Also, most available information focuses on the environmental characteristics of fire disasters. More emphasis on the epidemiologic characteristics of the host is needed so that the importance of such factors as behavior, knowledge, awareness, planning, perception, and predisposing medical factors can be determined more accurately. Arson specialists should be consulted to determine the need for epidemiologic assistance with the evaluation of incendiary fire disasters.

7. Prevention strategies need to encompass specific actions to address and minimize the risk of young children who are dependent on the knowledge and behavior of others.

8. More scientific information should be obtained that specifically addresses the adverse mental health impact on victims of and fire fighters involved with fire disasters.

9. More efforts should support the attempts by groups such as health departments, fire departments, and civic associations to determine the extent of and provision for smoke detectors in residential dwellings.

10. Fire-protection professionals should increase emphasis on public education and awareness of the proper selection, installation, usage, and maintenance of auxiliary heaters.

11. Public health and fire-protection professionals should stress that cigarette smoking is potentially dangerous—not only in terms of personal health—but also as a cause of fire disasters that destroy lives and property.

Summary

Contemporary fire disasters should be viewed as an unnecessary and preventable problem that deserves the attention and efforts of public health professionals—particularly in the United States, where the problems associated with fires are greater than in many other developed countries.

The literature contains limited statistics about the characteristics and adverse public health impact of fire disasters. However, these data still allow for identifying important contributions to fire disasters such as the role of residential fires, sources of ignition that include auxiliary heaters and cigarettes, and the need for widespread use of smoke detectors.

Appropriate public health prevention strategies appear to divide into five broad categories of activities: epidemiologic surveillance and research, engineering and legal controls, mitigation response and suppression, medical treatment and rehabilitation, and public awareness and education.

Additional efforts are needed in this field if the adverse public health impact of fire disasters is to be reduced further.

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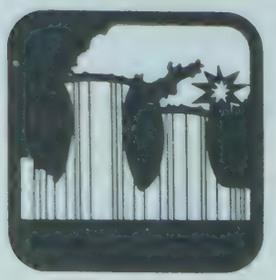

Nuclear-Reactor Incidents

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Background and Nature of Nuclear-Reactor Incidents

Introduction

Initial interest in nuclear fission centered on its potential as a weapon. However, by 1953 the United States Government determined that nuclear power should be developed as an energy resource, and the Atomic Energy Commission (AEC) began to promote the construction of reactors by private industry. By 1963, success had been marginal, and 23 reactors were wholly or partially owned by the Government. Private industry had begun work on seven reactors, but all were regarded as experimental or demonstration facilities. The use of nuclear power has increased since that time. However, in 1979 in the United States only 8% of the electricity was generated from nuclear power, compared with 18% in Switzerland and 15% in Germany (1). In 1983, 76 nuclear reactors operating commercially in 27 states produced about 12% of the electrical energy in the United States.

As the use of atomic power expanded, the risks associated with reactors became apparent. In the period 1950-1970, five U.S. nuclear reactors were damaged substantially by incidents. An incident at Idaho Falls SI-1 experimental reactor led to radiation exposure of the public, though at low levels. Exposure to the public from the incident at the Three Mile Island, Pennsylvania, reactor in 1979 was also minimal, but the damage to the reactor itself was severe. The incident at Chernobyl, in the Soviet Union, involved massive damage to the reactor core as well as substantial exposures to large numbers of people.

A radiologic incident can result in both acute, direct exposure of the public to the radioactive plume, as well as longer-term exposure to radionuclides deposited on food crops or in water. Similarly, the public health consequences of radiologic incidents can be acute or long-term. Acute health effects from radiation—such as vomiting and diarrhea—occur only in association with relatively high doses. In most cases, the long-term increase in the risk of cancer or genetic defects is of primary concern. Protection of the public from nuclear incidents begins with the design and siting of nuclear facilities and includes emergency-response planning and preparedness.

Characteristics of Nuclear Reactors

In any complex industrial process, incidents should be anticipated. A nuclear reactor has unique characteristics that pose special problems for emergency planners. To prevent or prepare for a radiologic incident, emergency planners must first understand how a nuclear reactor functions. In the reactor vessel or core, uranium (or, in some cases, thorium) nuclei are split by neutrons, and thermal energy is released. Two or more smaller atoms are created from the fission of each large uranium nucleus. Many of these new atoms are radioactive. These products of fission and the isotopes produced as they decay make up the core inventory. The fission process and the decaying of the products of fission generate heat that is removed by a coolant system (usually water) for conversion into steam and finally into electricity.

The uranium is packed inside fuel rods arranged in specific patterns within the core. During normal operation, fission products are trapped inside the fuel rods, but if heat builds up inside the core, the fuel cladding can melt and release fission products such as radioactive noble gases, iodines, cesium, and others. Fuel elements can also be damaged by chemical fires or explosions within the core. With commercial reactors, a containment building surrounds the core to provide physical containment of fission products and house mechanical safety features—including emergency cooling and filtration systems. These safety systems are designed to prevent a release of radioactive material into the environment. For example, filters can trap most large particulates and reactive compounds before they reach the environment. However, a release to the environment can occur if safety systems or the containment building are damaged by mechanical failure, human error, or a natural disaster such as an earthquake.

The Nuclear Fuel Cycle

Incidents can happen at any processing or transportation phase within the nuclear fuel cycle and during storage of spent fuel, so that environmental releases may involve a

variety of chemicals, uranium compounds, or fission products. The operation of the nuclear reactor is only one step in the fuel cycle. Uranium must first be mined and milled to produce yellow cake, a combination of all uranium isotopes. Since only certain isotopes can be fissioned in a reactor, the yellow cake is chemically processed and changed into a gaseous compound for enrichment. The fissionable isotopes are then separated and concentrated into a solid uranium oxide. Uranium oxide is shaped into pellets at a fuel fabrication facility and loaded into fuel rods. A fuel rod may last 3-4 years in a reactor before being removed to a waste-storage facility (2). Because the issue of permanent disposal of this high-level waste has not been resolved, spent fuel rods are usually stored on site in special tanks with coolant systems to prevent the buildup of heat from the decay of fission products. Any operation that requires movement of the fuel rods within or out of the core (or within the storage facility) increases the risk of an incident that could cause a release of fission products similar to those that would be released during an incident within the reactor core.

The processing of uranium involves a variety of hazardous chemicals, which could be released during an incident. For example, the incident in December 1985 at a uranium processing plant in Gore, Oklahoma, produced a cloud of hydrogen fluoride. One worker was killed and others were hospitalized from the acute effects of this vapor (3). Chemicals such as hydrazine and chlorine are used and stored at nuclear power facilities and could be released into water supplies or into the atmosphere during a fire or other incident. A partial list of the chemicals used at nuclear reactors and their potential health effects is shown in Table 1 (4,5).

TABLE 1. Chemicals used at nuclear power plants

Chemical	Health effects
Sulfuric acid	Irritating to eyes, skin, mucous membranes, and respiratory tract
Chlorine	Irritating to skin, eyes, mucous membranes; may cause nausea, vomiting, and acute respiratory distress
Ammonia	Irritating to skin, eyes, mucous membranes; may cause headache, burning of the throat, nausea, and vomiting
Sodium hydroxide	Corrosive to body tissues; may cause blindness, cutaneous burns, pulmonary irritation
Hydrazine	Irritating to eyes, upper respiratory tract; may cause skin burns, severe dermatitis, dizziness, nausea, animal carcinogen

Sources: References 4,5.

Public Health Consequences of Nuclear-Reactor Incidents

Routes of Exposure

A plume of airborne particulates and inert gases from the plant is the principal source of public exposure during most nuclear-reactor incidents. The first exposure pathways are external from being immersed in the plume or internal from inhaling gases or particulates. The plume is composed primarily of noble gases (e.g., krypton, xenon), organic, and inorganic iodides, and volatile inorganic material. The period of release may be short (a few hours) or last for over a month or more.

If the original release contains primarily inert radioactive gases, surface contamination and the resultant potential for long-term exposure will be minimal. However, if large quantities of particulates are released from the plant, surface contamination will be a substantial and long-term source of exposure. Occupants of a contaminated area could receive excessive whole-body or skin doses from radionuclides on the ground, cars, machinery, or on and within their own homes. Long after the initial release from the plant, public exposure from radionuclides deposited on the ground, food crops, or water can continue both by direct external radiation and through a number of ingestion pathways. Radionuclides can be ingested directly by drinking contaminated surface water or by eating fruits and vegetables coated by radioactive particulates. In addition, indirect pathways can develop through the food chain. For example, cows eating or drinking contaminated feed or water will produce milk containing radionuclides—most commonly iodine, strontium, or cesium. Food crops absorb radionuclides from the soil so that long-term contamination of locally grown fruits and vegetables or animal feed can be a serious health concern.

Potential Health Effects

ACUTE

A number of adverse health effects can be caused by a nuclear-reactor incident. Health effects can result from direct injury (e.g., burns associated with a break in a steam pipe), from the stress of the situation (e.g., myocardial infarction, psychological distress), or from exposure to radioactive material or chemicals released during the incident. Only radiation exposures that have adverse health effects are considered here because they are unique to radiologic incidents.

The biological effects of radiation exposure depend on the absorbed dose, the type of radiation, the rate of exposure and how much of the body and which organs are exposed (e.g., thyroid gland) (6). The absorbed dose is the amount of energy (measured in **rads** or **grays**) deposited in the body during radiation exposure. Different types of radiation (e.g., gamma rays, beta particles, alpha particles) produce different tissue damage at the same absorbed doses. Gamma radiation is electromagnetic radiation like ordinary light but is able to penetrate the body, and beta and alpha radiation are accelerated particles released by the nucleus during radioactive decay. A single radioisotope may emit more than one type of radiation. Because these types of radiation can affect tissues differently at the same dose, the absorbed dose is often multiplied by a quality factor to give what is called the dose equivalent (measured in **rems** or **sieverts**) so that exposures can be compared.

Exposure of part of the body such as an arm or leg or a single organ is less damaging than exposure of the whole body to the same dose. Dose rate is also a significant factor in determining the type of biological response. Because of

the body's repair mechanisms, the effects from a dose of 500 rem delivered instantaneously is quite different from those caused by the same dose given over a month or more. In general, adverse health effects increase with the combination of the total dose, the proportion of the body exposed, and the exposure rate.

Because genetic material is particularly sensitive to radiation, tissues that divide rapidly (e.g., blood-forming tissues, intestinal-lining cells) are more sensitive to damage than those that divide more slowly (e.g., muscles, nervous-system tissues). After an acute, whole-body dose of < 100 rems, an individual may have no outward symptoms, but may show increased chromosomal aberrations in blood lymphocytes and a decrease in blood count. A higher dose may produce acute radiation syndrome, an illness with dose-dependent symptoms. Acute, whole-body doses of > 100 rems may cause vomiting, hemorrhage, and an increased risk of infection due to reduced white-blood-cell counts. Treatment may include antibiotic therapy, blood transfusions, and possibly bone-marrow transplantation. Acute, whole-body doses of > 1,000 rems will damage the gastrointestional tract, provoking diarrhea and electrolyte imbalance, and may affect the central nervous system to cause seizures, gait disturbances, and coma. Ninety percent of persons exposed to such doses will die. Such acute, whole-body-radiation exposures in peacetime are very rare (7-9).

CHRONIC

More pertinent to most nuclear-reactor incidents are delayed effects from exposure to lower levels of radiation (10). Data on the biological effects of radiation have been collected from animal studies and studies of humans exposed to diagnostic (e.g., children exposed prenatally to abdominal X-ray examination of their mothers during pregnancy), therapeutic (e.g., treatment for ankylosing spondylitis), inadvertent occupational (e.g., radium-dial painters, uranium miners), and wartime irradiation (e.g., survivors of the atomic bombing of Hiroshima and Nagasaki in World War II). These studies provide evidence for three types of delayed effects: somatic effects for the exposed person, teratogenic effects for the fetus exposed in utero, and genetic effects for the offspring of the exposed person.

The main somatic effect of radiation exposures is cancer—especially leukemia and breast, thyroid, and lung cancer. According to current estimates of the risk from low-level radiation exposure, a whole-body dose of 1 rem increases an individual's lifetime risk of dying from any cancer by about 0.01% (10). The principal teratogenic effects described in studies of survivors of the atomic bombings of Nagasaki and Hiroshima have been mental retardation and reduced head size, especially persons who were exposed as fetuses 6-12 weeks post-conception. After 12 weeks of gestation, maternal exposure to significant quantities of radioactive iodine can destroy the fetal thyroid gland. Genetic effects among the offspring of the exposed population may include mutations. The estimated risk of mutations is about 1/1,000 live-born offspring/rem of parental exposure before conception, compared with a background rate of 107 gene mutations from other causes/1,000 live-born offspring.

None of these risk estimates are precise because they are extrapolated to low levels from relatively high radiation exposures. The exact risk at low levels of exposure is not known. Because the interaction of radiation with human tissue is believed to be harmful even at low levels, radiation exposures beyond natural background radiation and diagnostic or therapeutic exposures should be minimized.

Risk Factors for Exposure and Health Effects

The risk factors for exposure during a radiologic release are numerous. Obviously, both living adjacent to and downwind from a nuclear power plant increase an individual's chance of exposure if an incident occurs. Persons such as farmers or construction workers who work outdoors are also at additional risk because they probably take longer to return home for sheltering or evacuation. To alert these people in case of an incident, sirens should be used to supplement radio or television warnings. In addition, systems should be developed for alerting the hard of hearing. People who have difficulty evacuating are also at risk. For example, the handicapped, nursing-home and hospital patients, and prisoners require special aid and additional time to evacuate. In areas surrounding nuclear facilities, individuals who will need assistance should be identified as part of the local emergency-response planning. Plans should also be developed to alert local schools, hospitals, and prisons of any unintentional release. Some populations more sensitive to radiation exposure, such as children and fetuses, require extra consideration when protective actions are implemented. For example, children and pregnant women may be evacuated before the rest of the population or when lower exposure levels are expected. By developing county and state emergency response plans, additional risk to these special groups can be reduced.

Many of the risk factors for long-term environmental exposure after the plume has passed are identical to factors that can increase exposure during a radioactive release. Children and fetuses are more sensitive to radiation effects from external or internal exposure. Children are more vulnerable to exposure from radioisotopes in milk because they generally drink more milk than adults. People who live in rural areas and people of low economic status may eat more locally grown fruits and vegetables and are at greater risk of ingesting contamination. A population that uses surface water such as that from reservoirs or rivers may receive additional exposure from drinking water contaminated by surface runoff or by direct deposition from the plume.

Preventive and Control Measures

Design and Placement Factors

The prevention of a nuclear-reactor incident should be part of the planning stages of a plant. The choice of plant location involves geographic and meteorologic considerations. A site should not be selected in an area with high seismic activity (5), although it may not be possible to choose a site with no history of seismic vibrations. The probability of earthquakes can be estimated for a general area based on past seismic activity and the location of faults. The site should not be located in areas such as a flood plains or in tornado- or hurricane-prone areas. The potential health effects from a release can also be limited by locating the plant

in an area with a low population density and establishing an uninhabited area around the plant to act as a barrier between the reactor and the population.

After the site is selected, the plant should be designed in accord with the conditions at that particular site. Special construction features can increase the safety of the plant. In areas in which tornadoes or earthquakes may occur, the plant should be built to withstand high winds and impact from blowing debris in tornados or vibrations from minor earthquakes.

Factors Relating to Plant Operation

Despite the fact that many of a nuclear reactor's safety systems are computer-controlled, operators are essential to the safe operation of the plant. To lower the probability of human error, plant personnel are trained to respond to unusual conditions within the plant and are assigned specific responsibilities during an incident. However, fatigue from rotating shifts, boredom, and inadequate training or supervision can lead to serious human error. In fact, all radiologic incidents can be partially attributed to human error. To deter deliberate sabotage of the reactor, security systems can be used to prevent unauthorized personnel from being on site and to limit access to sensitive areas of the plant.

Offsite, elected officials and emergency workers such as fire fighters and policemen should also learn to assist in activities such as evacuation and to protect themselves and others from radiation hazards. State and Federal emergency-response plans provide a blueprint for agencies to respond to emergencies and minimize—to the extent possible—errors in human judgment during an incident.

Reducing Off-Site Exposures During a Radiologic Release

The U.S. Environmental Protection Agency (EPA) has set "protective action guides," which are levels at which action should be taken to lower the potential radiation exposure of the public. These doses are not actual exposures but are projected or estimated doses if no action is taken. For the general population, EPA recommends that protective action be taken if the projected thyroid dose is 5-25 rem or if the whole-body dose is 1-5 rem (11). However, more stringent limits may be applied by state health authorities, particularly for pregnant women and children. Protective actions may include evacuation, sheltering, or respiratory protection in addition to, or in place of, administering potassium iodide (KI).

In the event of a nuclear-power-plant incident, actions to minimize or eliminate exposure of the public begin in the period between the realization that a potentially serious problem is developing and the actual release of radioactive material. If a safety system or other component of a reactor is damaged or malfunctions, Federal emergency-response plans require that local, state, and Federal officials be notified. The degree of response by state and Federal agencies depends on the severity of the incident and the size of the potentially exposed population. Federal and state activity increases dramatically if a release is imminent. Decisions to initiate a particular protective action are based on factors such as local weather conditions as well as the conditions at the plant, which determine the probability of a release and which isotopes will be released. To be effective, all protective actions should meet the following criteria (9).

- The action must be effective in reducing or preventing exposure to the public and must not carry health risks greater than those of the incident itself.
- The implementation must be feasible both logistically and financially.
- The agency or agencies responsible for implementing the protective action must be clearly identified, and the authority to implement the action must exist.
- The action must not have a large economic impact on the public, business, industry, or government compared with the health and economic impacts of the incident.

IMMEDIATE PROTECTIVE ACTION

In the early stages of an incident, a number of actions can be implemented to protect the public. One of the first decisions to be made is whether to advise evacuation or whether to advise residents to remain in their homes with windows and doors shut and ventilation turned off while the radioactive plume passes. Together, sheltering and respiratory protection in the form of wet handkerchiefs or towels can lower the inhalation of particulates but not of noble gases (12). Sheltering can also reduce gamma exposure from the plume by a factor of 2-10 (12), but it is a viable alternative only for short periods due to the infiltration of gases and vapors into the dwelling by normal air exchange with outside air. The use of sheltering is questionable if the release period is unpredictable or likely to be longer than several hours. Evacuation is a more costly but generally more effective method for reducing public exposure before a release has occurred. The decision to evacuate must include considerations such as weather conditions (e.g., the presence of a blizzard may make evacuation an unsuitable alternative), the likelihood of a release, the availability of shelters for the evacuees, and the quality of the evacuation routes. If a release has already begun, the benefits of evacuation must be weighed against the increased dose accrued while evacuating.

If radioiodine is released from the plant, the administration of stable iodide in the form of potassium iodide (KI) can lower or block the uptake of radioiodine by the thyroid. However, iodide will not protect against external radiation exposure or exposure to other inhaled radionuclides. To be effective, the iodine must be administered before or shortly after (within 1 day) exposure to radioiodine (11,12). Although some persons may also suffer side effects after taking KI, a risk assessment by the Food and Drug Administration (FDA) indicates that the risk from a projected thyroid dose of $\geq$ 25 rem outweighs the risk from short-term use of KI. During an actual release, the potential dose of radioiodine to the thyroid is estimated using dispersion modeling and the actual conditions at the plant. The decision to use or not use KI and how it should be distributed is left to the individual states (13). However, the rapid distribution of KI tablets required during an emergency is difficult, stockpiling for an unlikely release is costly, and the KI tablets have a limited shelf-life.

LONG-TERM PROTECTIVE ACTIONS

If the external exposure rate from surface contamination is high, residents can be evacuated or, if already evacuated, permanently relocated outside the contaminated area. Addi-

tionally, access to highly contaminated areas can be physically restricted to prevent the public from entering. In less severe situations, dilution and removal of contamination can be attempted by washing cars, houses, and streets and by trimming grass and disposing of the clippings (12). Weathering from rain or snow also decreases the concentration of radioisotopes on structures and on the ground surface, although surface runoff may recontaminate local lakes and streams after each heavy rain.

Direct ingestion of contamination can be prevented by supplying fresh drinking water to residents if necessary. Normal food preparation such as peeling or washing can remove the contamination on some fruits and vegetables (13). Food that cannot be appropriately decontaminated may need to be destroyed. Significant contamination of milk can be avoided by providing uncontaminated feed and water to cattle. The success of this action depends on the availability of stored feed and fresh water and the ability of farmers to remove cows from pastures in a short time.

FDA has developed action levels based on the projected doses from food or milk at which protective or emergency actions would be appropriate. According to these guidelines, if the projected dose to the public from food consumption is 0.5 rem whole-body or 1.5 rem to the thyroid, cattle should be given stored feed instead of being allowed to graze. At a projected dose of 5 rem whole body or 15 rem to the thyroid, responsible agencies are expected to prevent the food or milk from entering into commerce either by storage for decay or by condemnation (14). For example, milk contaminated with short-lived isotopes can be diverted into milk products such as cheese. However, storage is not practical if the food or milk is contaminated with long-lived radioisotopes. For local food crops, long-term recommendations must be based on soil concentrations of each radioisotope and on the effectiveness of soil management and decontamination efforts (15).

Reducing the Adverse Health Impact After Exposure

Acute

If, despite precautions to prevent exposure, some persons are exposed through a nuclear-plant incident to external irradiation or internal sources of radiation, morbidity and mortality from these exposures can still be prevented (8,9). In the case of acute exposures, emergency lifesaving assistance to prevent shock from trauma or to maintain respiration has highest priority. Persons exposed to external X-ray, gamma, or other radiation may require symptomatic treatment at a specialized hospital if their whole body dose is > 50 rem. People whose skin or clothing has been contaminated by radioactive material may pose a hazard not only to themselves but also to the hospital environment, staff, and other patients. These people must go through decontamination procedures to avoid adverse health effects and to protect other people from being exposed (16).

Treatment for radiation injuries depends on the degree of exposure and on whether the exposure is internal or external (9,17). It is extremely unlikely that a live patient will be so contaminated as to pose an acute radiation risk to rescue or medical personnel. Therefore, for any acutely exposed radiation victims, the usual priorities of emergency care—the saving of life and the prevention of further injury—take precedence over decontaminating the patient or minimizing exposure of attending personnel.

Assessment of the level of exposure of a hospitalized individual (e.g., a heavily exposed worker) is generally more accurate than assessment of exposure of the general population. Personal dosimeters; direct measurement of radioactivity in and on the body; and clinical assessment of symptoms, signs, and white blood cell counts may provide evidence of the severity of exposure. For the general population, exposures can be estimated from levels of radiation measured by detectors around the plant and factors such as distance and direction from the plant and time spent at different exposed locations (18). Analysis of biological samples as well as whole-body radiation counters can be used to detect internal levels of radioisotopes. Even though studies of chromosomal aberrations in blood lymphocytes may detect exposures as low as 10 rem within a few hours after an incident (9), medical examinations and treatment should be confined to individuals who are highly exposed, or contaminated, or have ingested or inhaled significant quantities of radioactive material.

Chronic

For low-level chronic exposures, both internal and external, it is unclear whether long-term follow-up and monitoring can reduce subsequent morbidity and mortality. Epidemiologic studies based on registries of exposed persons, such as the Three Mile Island Population Registry (18), may provide further information on the effects of low-level radiation, although their low statistical power may make interpretation difficult (19).

History of Nuclear-Reactor Incidents

Many of the major nuclear reactor incidents in the United States have involved test reactors or experimental breeder reactors that create plutonium-238 during the fission process. Four of these incidents did not cause a release of radiologic material to the environment despite damage to the core. These incidents involved the Chalk River Reactor; the Idaho Experimental Breeder, Unit 1; the Westinghouse Test Reactor; and Detroit Edison's Fermi Reactor (breeder). Significant quantities of radioiodine were released during two reactor incidents at facilities in the United States and one in England. Of these, the Windscale reactor in England and the SL-1 reactor in the United States did not have containment buildings and were not for commercial use or power production. However, the incident at Three Mile Island—a commercial reactor—resulted in substantial damage to the core and the release of radioactive noble gases and radioiodine (9). The more recent incident at Chernobyl in the Soviet Union is the most serious incident recorded at any nuclear-power facility, causing massive damage to the core and allowing millions of curies of fission products to escape into the environment. A summary of these incidents is given in Table 2. Descriptions of some of the incidents are presented below.

TABLE 2. Incidents with core damage in nuclear reactors

Description of incident	Site	Date	Adult thyroid dose (rem)
Minor core damage (no release of radiologic material)	Chalk River	1952	NA*
	Breeder Reactor, Idaho	1955	NA
	Westinghouse Test Reactor	1960	NA
	Detroit Edison Fermi	1966	NA
Major core damage (radioiodine released)			
Noncommercial	Windscale, England	1957	16
	Idaho Falls SL-1	1961	0.035
Commercial	Three Mile Island	1979	0.005
	Chernobyl, Soviet Union	1986	100†

* Not applicable.

† Estimated median dose.

Chernobyl, Soviet Union

Elevated levels of environmental radiation were detected during routine monitoring at a nuclear-power facility in Eastern Sweden on April 28, 1986. The radioactive cloud directed attention eastward into the Soviet Union (20). Shortly afterward, the Russian government reported that an incident had occurred at reactor number four in Chernobyl located in the Ukraine. In fact, on April 26 at 1:23 a.m., an explosion tore open the reactor core, dispersing an estimated 12 million curies of radioisotopes into the environment during the first 24 hours after the incident. Over the next 10 days, another 38 million curies escaped from the burning graphite core (21).

The reactor was one of the many graphite-moderated reactors that produce approximately 4% of the electrical power in the Soviet Union (22). Though construction features of this reactor are unique to Russian graphite-moderated reactors, the basic cause of the incident—human error—is not. The incident started during a test procedure, when critical safety systems were shut down, allowing the reactor to surge out of control in a matter of minutes. Reports on May 2 indicated that all graphite reactors (16-20) had been shut down temporarily because of the incident.

The first reports from the Soviet Union indicated that two workers were killed immediately and that 197 were injured—18 seriously. Immediately after the incident, 1,000-2,000 people were screened for signs of acute radiation exposure, and 400-500 were admitted to hospitals—although only 200 had indications of radiation exposure. Of the persons hospitalized, 80 developed acute radiation syndrome. By June 7, 1987, 27 deaths had been attributed to the incident (23).

Evacuation of the general populace was not begun until April 27, 1986, when at least 49,000 people were relocated. The delay is attributed to administrative issues and a lack of understanding about the seriousness of the incident. A total of 135,000 people were eventually evacuated from the area. By June 1987 two towns within an 18-km area had been reinhabited, and 16 other areas were being prepared for habitation. However, 27 towns have been permanently abandoned because of high levels of contamination (24). Soviet reports indicate that people within 15 km of the plant received doses of 35-50 rem.

Fallout from the incident spread over large parts of Europe during the release. Large variations in the deposition of radionuclides due to rain created localized hot spots. Long-term exposures will continue through the food chain and direct irradiation from deposited radionuclides. The number of future deaths from cancer attributable to radiation exposure can only be speculated about at this time because of the assumptions needed to estimate the total population dose. Although estimates have ranged from 5,000 to 40,000 cancer deaths over the next 70 years, 9.5 million individuals are expected to die of cancer from other causes in the same time period (21). A 2% and 0.001% increase was predicted for teratogenic effects and genetic abnormalities, respectively, affecting babies born within 9 months after the incident (25). However, a study of nervous-system and eye defects in Western Europe indicated an increase in numbers of neural-tube

defects only in Odense, Denmark, and no general increase in congenital nervous-system or eye defects (26).

A number of European countries took protective action after the incident. Poland banned the sale of milk from pastured cows and issued potassium iodide to 11 million children. In Romania, children were kept indoors, and the government recommended that fruits and vegetables be washed before being consumed. Without additional information it is difficult to say whether actions taken in these and other European countries were useful in lowering exposures. Some actions, such as distribution of potassium iodide, may have been taken too late to be effective or may have been unnecessary (27). Exposure rates as high as 100 milliroentgen/hour were reported in Belgrade, Yugoslavia. However, without knowledge of how widespread these levels were or how long they existed, the adverse health impact on the population cannot be predicted.

Some Americans who were in Russia during the incident were monitored for radiation when they returned to the United States. Thyroid burdens for these people ranged from undetectable quantities to 270 nanocuries of iodine^{131}, which would result in a dose of about 2 rem to the thyroid (28). Exposures in the United States from the fallout were negligible.

Three Mile Island, Pennsylvania

On March 28, 1978, at the Three Mile Island power plant near Middletown, Pennsylvania, a series of mechanical failures and human errors led to a loss of coolant in the Unit 2 reactor, which allowed the fuel to overheat. During the incident, contaminated coolant water was routed out of the containment building into an auxiliary building. Volatile radionuclides escaped through the ventilation system, after passing through a filtration system that removed the chemically active compounds—including most of the radioiodine (2). The principal radionuclides released were xenon and small quantities of iodine (29). During the incident, Pennsylvania Governor Richard Thornburgh advised all people within 10 miles of the plant to remain indoors and recommended that all pregnant women and preschool-age children within 5 miles of the plant evacuate. However, reports after the incident by an interagency dose-assessment group indicated that the largest external dose was < 100 mrem, and the average dose was 1.5 mrem. Health surveys indicate that the most significant health impact from the incident was psychological due to the stress of the incident— not an impact of exposure to radiation (30,31). On the basis of dose calculations, one to two individuals in the exposed population would be expected to develop either cancer or genetic effects from the radiation exposure (4).

Soon after the Three Mile Island incident, the Pennsylvania Department of Health (PDH), with assistance from CDC and the U.S. Bureau of Census, set up a population registry of the nearly 36,000 persons who lived within 5 miles of the plant (32). Each registrant provided information about total time spent in the 5-mile area during the 10 days after the incident so that individual radiation doses could be estimated. Demographic and health-related information was also gathered to use in future epidemiologic studies of adverse health effects. Among women registrants who said they were pregnant at the time of the incident, the estimated incidence of miscarriage before completion of 16 weeks of gestation or of delivery of a dead fetus after 16 weeks of gestation resembled those reported elsewhere in non-exposed populations (33).

The PDH has also collected information on some 8,000 women who lived within 10 miles of the plant and who gave birth within 2 years of the incident, and is conducting studies of cancer incidence and mortality among nearby residents. Persons within 20 miles of the plant reported feeling distressed after the incident, but this dissipated within a month; distrust toward authority, however, persisted much longer (34). Planning responses for emergencies (e.g., centralizing public information, controlling rumors) may reduce this distrust during future incidents (35).

Windscale, England

When the United Kingdom began production of nuclear weapons, plutonium-producing reactors were constructed at a site on the Northwest coast of England called Windscale. In 1957 a fire started in one reactor due to overheating, and a substantial amount of fission products was released. Estimates of the whole-body gamma dose to residents nearby range from 30 to 75 mrem (34). Radioiodine was released during the incident, and protective actions for milk were implemented. The highest thyroid dose was 16 rad for children and 4 rad for adults (35,36). Other food products such as vegetables, meats, and water did not require protective action. Ingestion of radioiodine in milk was considered the most important pathway of exposure.

There is considerable uncertainty about the health impact of radiation released during the Windscale incident and afterward through normal effluents (36-39). More study is needed before conclusions can be drawn about cancer or other health effects from the Windscale incident.

Cheliabinsk, Soviet Union

In 1957, a major incident occurred at a reprocessing facility in the Soviet Union. Although this incident did not occur at a nuclear power plant, the results are typical of what might be expected from a catastrophic incident at a nuclear plant. Details have not been directly reported from the Soviet Union; however, theories about the nature of the incident have been proposed on the basis of reviews of radioecology literature. The release involved a military facility near Kasli in the Cheliabinsk Province. Evidence suggests that improper storage and chemical processing may have caused a single release or a series of radioactive releases. Contamination was extensive and may have resulted in the permanent relocating of residents from an area of 100-1,000 square kilometers. Contamination of a principal river and several lakes and reservoirs was severe enough to warrant relocating the entire populations of several towns (40). Because of the secrecy in the Soviet Union surrounding this incident, little is known in the United States about associated acute or chronic health effects.

Planning for the Future

Probability of Future Incidents

In 1957, the Atomic Energy Commission completed an evaluation of the possible health consequences of an

unintentional release of radioisotopes from a nuclear power plant. However, this evaluation did not assess the probability of a particular incident or the effectiveness of engineering safety features built into plants (2). A more thorough study completed in 1975 (41) provided a quantitative and realistic evaluation of the risks to the public from nuclear-reactor incidents, developed methodologies for assessing these risks, evaluated the status of reactor safety, and identified areas requiring additional research. The study addressed only the 100 light-water reactors expected to be in operation in the early 1980s (light-water reactors are the most common commercial reactors, although other types are in operation). This report excludes boiling-water reactors, as well as other commercial reactors, weapons-producing facilities, and reactors in other countries.

For purposes of risk assessment, possible health effects from a release were divided into five categories: all fatalities within 1 year, all fatalities from cancer, injuries requiring treatment, thyroid injuries requiring treatment, and genetic effects (8). This report predicted that the probability of an incident that could cause approximately one of each of these health effects was 1 in 200/year/100 operating reactors. The most serious incident evaluated in the report would cause 1,500 cases of cancer/year in the 10-40 years after an incident. The probability of this type of incident was calculated to be 1 in 10 million/year (2,18). The potential error in this type of prediction can be several orders of magnitude since the sequences of incidents and the success of engineering safeguards must be predicted by mathematical models whose accuracy cannot be tested.

Surveillance for Incidents

The Nuclear Regulatory Commission (NRC) and the Federal Emergency Management Agency (FEMA) have established a system to identify unusual occurrences at nuclear facilities. The operator of a nuclear power facility must notify the NRC of changes in the plant's normal status so that officials can prepare to respond immediately to any release that occurs (9). A tiered classification system based on four emergency action levels defines the severity of the status of a reactor and the potential for a release. Investigation of reports of unusual occurrences can clarify the types of incidents that occur at a nuclear facility. Other nuclear-power-plant operators are notified of the results from these investigations so that they can evaluate the safety of their own procedures and avoid similar incidents. The emergency action levels defined by NRC are described in Table 3.

Emergency-Response Planning

Since the Three Mile Island incident in 1979, FEMA has developed a national contingency plan, the Federal Radiological Emergency Response Plan (FRERP), to coordinate Federal response to peacetime radiologic emergencies (42). The FRERP describes the Federal government's concept of operations for responding to radiologic emergencies, outlines Federal policies and planning assumptions that underlie this concept of operations and on which Federal agency response plans (in addition to their agency-specific policies) are based, and specifies authorities and responsibilities of each Federal agency that may play a substantial role in dealing with such emergencies (43).

Individual Federal agencies (e.g., Department of Health and Human Services, Centers for Disease Control) have developed their own more specific plans applicable to their unique capabilities and responsibilities (43). All operating nuclear-power-plant sites have state and local offsite emergency-preparedness plans, but they have not all been approved by FEMA. The General Accounting Office has reported to the U.S. Congress on ". . .further actions needed to improve emergency preparedness around nuclear power plants," especially the need for better centralized Federal agency control and coordination (44).

"Tabletop" and field exercises are regularly conducted by FEMA to test Federal plans for radiologic emergency response. All agencies with primary responsibility participate in these exercises to test their own readiness and to refine the Federal response plan. State and local officials also participate in these FEMA-sponsored exercises so that methods for local, state, and Federal interactions can be developed. However, no exercise can fully test an emergency-response plan or can fully anticipate political, economic, or social issues that may drive public health recommendations during an emergency.

TABLE 3. Emergency action levels defined by the Nuclear Regulatory Commission

Emergency action level	Plant status
Notification of unusual event	Potential degradation of the normal level of plant safety with no release of radioactivity requiring offsite response
Alert	Actual or potential degradation of plant safety at a substantial level; any potential release expected to be well below established emergency action levels
Site-area emergency	Actual or probable failure of safety systems that normally provide protection for the public; potential releases not expected to exceed established action levels except at plant boundary
General emergency	Actual or imminent core degradation or melt down with a potential for loss of containment protection; potential releases expected to exceed established action levels

Recommendations

1. Education about the potential risks from nuclear reactors, the effects of any radiologic release, and protective actions that can be taken in the event of a release may alleviate the mental stress from a future incident and minimize inappropriate or unnecessary action by the public.

2. More research is needed on ways to control human error associated with radiologic incidents. Appropriate questions include: Should safety systems be designed so that plant operators cannot turn them off? Should rotating shifts be eliminated? Do current training programs need improvement? How can quality work be maintained during off-shifts such as weekends or nights?

3. Since incidents can occur throughout the nuclear fuel cycle, Federal emergency-response planning should place more emphasis on planning for incidents at all parts of the cycle rather than concentrating efforts solely on preparation for a catastrophic event at a nuclear power reactor.

4. Additional research is needed to determine which isotopes will most likely pose the greatest source of exposure during a variety of release scenarios. Until the TMI incident, emergency plans for reactors centered around a large release of radioiodine. However, during the TMI incident, significantly less radioiodine was released than expected; and at Chernobyl, long-lived isotopes pose a more significant hazard than would have been estimated previously.

5. There is a continuing need within state and Federal agencies for expertise in emergency response and radiologic safety. Methods for building current resources need to be investigated.

6. More concrete guidance to states on the efficiency of stockpiling potassium iodide or distributing it during an incident is needed.

7. Traditional methods for treating victims of a major radiologic incident need to be re-evaluated in view of the experience gained during the treatment for trauma or severe radiation exposure at Chernobyl.

Summary and Conclusions

The reactor core is the central component of a nuclear power plant. However, complex mechanical systems cool and protect the reactor, convert the thermal energy to electricity, and filter effluents. Natural disasters, mechanical failures, and human errors can all contribute toward an incident by damaging the safety systems or the core itself. A release of radioactive material such as noble gases and radioiodine is most likely to be caused by a series of malfunctions or errors rather than by a single event. To prevent exposures, nuclear power plants can be designed to minimize the possibility of an incident caused by a natural disaster or mechanical failure. Plant personnel can also be trained to maintain safety systems within the plant and to response appropriately if an incident does occur.

Health effects from a radiologic release can be acute or long-term. Relatively high doses of radiation can damage the bone marrow, intestinal lining, and the nervous system depending on the magnitude of the exposure. Cancer or genetic defects induced by radiation exposure may not appear until many years after exposure and may be induced by low levels of exposure. Chemicals stored on-site at nuclear facilities can also pose a health hazard during an incident. To prevent adverse health effects from an incident at a nuclear facility, exposure to the public can be avoided or reduced when an incident occurs. The public around a nuclear plant can be evacuated or sheltered before or during an unintentional release to prevent external exposure and inhalation of radioisotopes. After the release has ended, food and water pathways as well as surface contamination can be important sources of exposure. Supplying fresh food and water can minimize direct ingestion of radioisotopes. However, radionuclides can also build up in food chains (e.g., the cow to milk to human pathway) and may require different strategies to prevent exposure. If exposure occurs despite protective actions, morbidity and mortality can be reduced through medical care for acute effects and possibly through long-term screening for cancer.

Populations and individuals that are more sensitive to radiation may be at higher risk from an incident. Children and fetuses are more sensitive to radiation effects than adults and are more likely to be exposed through the cow-milk pathway than adults. Individuals living closest to a reactor are at higher risk of exposure during an incident, as are persons who work outdoors. Individuals who eat vegetables and fruit from local gardens are more likely to ingest radionuclides through food pathways. The elderly, handicapped, or hospitalized require special assistance during an emergency. Protective actions to reduce the risk for sensitive populations and other individuals from a radiologic release should be addressed in radiologic emergency-response plans.

Although major incidents at commercial nuclear plants are rare, several have occurred. In 1957, a fire at the Windscale, England, facility caused a release that led to protective actions taken for milk but not agricultural products. An extremely severe incident reportedly occurred at a reprocessing facility in the Soviet Union in 1957, although very little is known about its cause or about the adverse health effects on the surrounding population. The Three Mile Island incident in 1978 has been the most significant nuclear reactor incident in the United States in terms of potential public exposure. However, actual doses to the public were calculated to be <100 mrem, and the most significant adverse health effect was stress on the population around the plant.

Federal and state planning for nuclear-power incidents was expanded after the incident at TMI. State emergency plans and exercises to test those plans are required around the nuclear facilities. A Federal plan has also been developed and tested through exercises. However, in both state and Federal plans, exercises are not likely to fully explore political, economic, social, and technical problems that will develop during an actual emergency. In light of this fact, flexibility must be allowed within emergency-response plans, and the technical and managerial expertise to cope with emergency-response issues must be maintained within state and Federal agencies.

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